A STUDY ON ACUTE SYMPTOMATIC SEIZURES IN THE ELDERLY

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

in partial fulfilment of the requirements
for the award of the degree of

DM (NEUROLOGY) – BRANCH – I

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

AUGUST 2011

CERTIFICATE

This is to certify that the Dissertation entitled, **"A STUDY ON ACUTE SYMPTOMATIC SEIZURES IN THE ELDERLY"** is the bonafide record work done by Dr.D.Muthukumaran, under our guidance and supervision in the Institute of Neurology, Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai, submitted as partial fulfilment for the requirements of D.M. Degree examination Branch I NEUROLOGY, AUGUST 2011, under The Dr.M.G.R. Medical University, Chennai.

DR.R.M.BHOOPATHY .DM.
PROFESSOR OF NEUROLOGY,
INSTITUTE OF NEUROLOGY,
MADRAS MEDICAL COLLEGE,
CHENNAI-3

DR.V.SUNDAR. Mch.
PROFESSOR AND HEAD OF THE DEPARTMENT,
INSTITUTE OF NEUROLOGY,
MADRAS MEDICAL COLLEGE,
CHENNAI-3

Dr.V.KANAGASABAI, M.D.,
THE DEAN,
MADRAS MEDICAL COLLEGE,
CHENNAI-3.

DECLARATION

I solemnly declare that this dissertation titled "**A STUDY ON ACUTE SYMPTOMATIC SEIZURES IN THE ELDERLY"** is done by me in the Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of Prof. Dr.R.M. Bhoopathy, M.D.,D.M., Professor of Neurology, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr.M.G.R Medical University, Chennai in partial fulfilment of the university requirements for the award of the degree of D.M. Neurology.

Place : Chennai
Date :

Dr.D.Muthukumaran.
D.M.Postgraduate student,
Institute of Neurology,
Madras medical college,
Chennai.

ACKNOWLEDGEMENT

It gives me great pleasure to acknowledge all those who guided, encouraged and supported me in the successful completion of my dissertation.

First and foremost, I express my gratitude to, the Dean Dr.V.Kanagasabai M.D.for having permitted me to carry out this dissertation work at Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai.

I am extremely thankful to Prof. Dr.V.Sundar.M.ch., Professor of Neurosurgery, Head of the department, Institute of Neurology, Rajiv Gandhi Government General Hospital Chennai for his constant encouragement, valuable guidance and support.

I express my deep sense of gratitude and sincere thanks to our respected and beloved Chief Dr. R.M. Bhoopathy. M.D., D.M., Professor of Neurology, Institute of Neurology, Rajiv Gandhi Government General Hospital Chennai for his teaching, valuable suggestions, constant motivation, kind guidance and moral support in the conduct of this study.

I express my sincere thanks and gratitude to our Professors Dr.C.Mutharasu.D.M., Dr.K.Bhanu.D.M., Dr.R.LakshmiNarasimhan.D.M, Dr. S. Balasubramanian. D.M., for their valuable suggestions and support.

I express my sincere thanks to Prof. Dr. C. Rajendiran M.D., Director of Institute of General Medicine, Rajiv Gandhi Government General Hospital Chennai for allowing me to collect the case materials and for his guidance.

I am extremely thankful to our Assistant Professors Dr.V.Kamaraj. D.M., Dr.S.Arunan..D.M.,Dr.M.Jawahar.D.M.,Dr.P.Muthukumar.D.M., Dr. V.Kannan.D.M., for their valuable guidance and support.

I owe my sincere thanks to all those patients and the technical staffs who participated in the study for it is their cooperation which made this study possible.

CONTENTS

LIST OF ABBREVIATIONS

CNS	-	Central nervous system
CT	-	Computerized Tomogram
MRI	-	Magnetic Resonance Imaging
EEG	-	Electro Encephalogram
ILAE	-	International League Against Epilepsy
GTCS	-	Generalized Tonic Clonic Seizure
AED	-	Anti Epileptic Drug
CSF	-	Cerebrospinal Fluid
HIV	-	Human immunodeficiency virus
FND	-	Focal neurological deficit
ICH	-	Intracerebral haemorrhage
CVT	-	Cerebral venous thrombosis
GRE	-	Gradient echo sequence
DWI	-	Diffusion weighted image

INTRODUCTION

Acute symptomatic seizures[1] are those caused or provoked by an acute medical or neurological insult and it is as common as epilepsy in the medical wards. Acute symptomatic seizures differ from epilepsy in several important aspects. First, unlike epilepsy, these seizures have a clearly identifiable proximate cause, to the extent that one can never be certain of a causal association. When one considers the temporal sequence of acute symptomatic seizures (e.g.uremia, head injury, or stroke immediately preceding a seizure), the biologic plausibility (acute disruption of brain integrity or metabolic homeostasis) and in many cases the dose effect (severity of injury correlated with the risk for seizures) all quite compellingly indicate causation. Although a risk ratio for the immediate association between cause and effect has not been calculated, it must be enormous. Second, unlike epilepsy, acute symptomatic seizures are not characterized by a tendency to recur. The risk for subsequent epilepsy may be increased in individuals experiencing such insults; in general, one does not expect seizures to recur unless the underlying condition recurs. As a corollary, such individuals usually do not need to be treated with antiseizure medication on a long-term basis, although such treatment may be warranted on a short-term basis until the acute condition is resolved[2]. It may be single or repetitive. Type of seizure can be focal with or without generalisation or generalised. Most common is generalised tonic clonic

seizure. Non convulsive seizure and status are common in patients admitted to intensive care units. Partial seizure is usually associated with structural abnormalities of brain but generalised seizure may result from cellular or structural abnormalities that have a wide spread distribution. In developing countries most common cause of acute symptomatic seizure is CNS infections. Seizures in the elderly may be caused by stroke, systemic metabolic conditions, subdural hematoma, central nervous system infection, degenerative disorders, or malignancy. The etiology of seizures is multifactorial in any given individual and is best thought of as an interaction between genetically determined seizure thresholds, underlying predisposing pathologies or metabolic derangements and acute precipitating factors. Very few studies are there, analysing the causes of acute symptomatic seizures, in the elderly patients . So it is useful to study the various conditions producing seizures in our patients and the use of investigations to find out the underlying problem. A detailed medical history, a thorough physical examination, especially of the nervous system, analysis of blood and other body fluids, electroencephalographic (EEG) recordings, magnetic resonance imaging (MRI) and/or computerized tomography (CT) scans we are able to find out the underlying cause of acute symptomatic seizures. Accurate diagnosis of the cause of acute symptomatic seizure is very important in the treatment and prognosis as the treatment of underlying condition will abolish the seizure .

REVIEW OF LITERATURE

A seizure is a paroxysmal event due to abnormal, excessive, hypersynchronous discharges from an aggregate of central nervous system neurons. Depending on the distribution of discharges, this abnormal CNS activity can have various manifestations, ranging from dramatic convulsive activity to experiential phenomena not readily discernible by an observer. Although a variety of factors influence the incidence and prevalence of seizures , 5–10% of the population will have at least one seizure, with the highest incidence occurring in early childhood and late adulthood[3,4].

Epilepsy describes a condition in which a person has recurrent seizures due to a chronic , underlying process . This definition implies that a person with a single seizure , or recurrent seizures due to correctable or avoidable circumstances, does not necessarily have epilepsy. Epilepsy refers to a clinical phenomenon rather than a single disease entity , since there are many forms and causes of epilepsy . However, among the many causes of epilepsy there are various epilepsy syndromes in which the clinical and pathologic characteristics are distinctive and suggest a specific underlying etiology.

Definitions of Terms [5]

A non epileptic event is a clinical event presumed to be unrelated to abnormal and excessive neuronal discharge. An example of a non epileptic event is syncope.

An epileptic seizure is a clinical event presumed to result from an abnormal and excessive neuronal discharge. The clinical symptoms are paroxysmal and may include impaired consciousness and motor, sensory, autonomic, or psychic events perceived by the subject or an observer.

A provoked seizure is an acute symptomatic seizure that occurs following a recent acute disorder such as a metabolic insult, toxic insult, CNS infection, stroke, brain trauma, cerebral haemorrhage, medication toxicity, alcohol withdrawal, or drug withdrawal.

An unprovoked seizure is a cryptogenic or a remote symptomatic seizure.

Epilepsy occurs when 2 or more epileptic seizures occur unprovoked by any immediately identifiable cause. The seizures must occur more than 24 hours apart. In epidemiologic studies, an episode of status epilepticus is considered a single seizure. Febrile seizures and neonatal seizures are excluded from this category.

Frequency:

A first seizure is a sudden frightening event for the individual, onlookers and family members. Available data on an individual's lifetime risk of developing one episode of non-febrile seizures is at least 4% .[6] A first seizure caused by an acute disturbance in brain function (acute symptomatic or provoked) is unlikely to recur (3 to 10 %).[7] If a seizure is unprovoked, however, meta-analysis suggests that 30 to 50 % will recur; and after a second unprovoked seizure, 70 to 80 % recur, justifying the diagnosis of epilepsy. [8]

Sex:

Most authors report a small-to-moderate increased preponderance in men in their studies of first seizures in adults[9]

Age [10]

In practice it is useful to consider the etiologies of seizures based on age of the patient, as age is one of the most important factors determining both the incidence and likely causes of seizures.

Causes for seizures in Neonates:

Perinatal hypoxia and ischemia, intracranial haemorrhage and trauma, acute CNS infection, metabolic disturbances, drug withdrawal, genetic disorders.

Causes for seizures in Infants and Children:

- Febrile seizures, genetic disorders, developmental disorders, trauma., Idiopathic.

Causes for seizures in Adolescents:

- Trauma.
- Genetic Disorders.
- Infection.
- Tumor.
- Illicit drugs.
- Idiopathic

Causes for seizures in young adults:

- Trauma.
- Alcohol Withdrawal.
- Illicit drug use.
- Tumor.
- Idiopathic.

Causes for seizures in older adults:

- Cerebrovascular disease
- Brain tumor
- Alcohol withdrawal
- Metabolic disorders
- Degenerative diseases
- Idiopathic

CLASSIFICATION OF SEIZURES

Seizures have been classified in several ways: according to their supposed etiology, i.e., idiopathic (primary) or symptomatic (secondary); their site of origin; their clinical form (generalized or focal); their frequency (isolated, cyclic, or repetitive, or the closely spaced sequence of status epilepticus); or their electrophysiologic correlates. The classification of seizure was first proposed by Gastaut in 1970 and was then refined repeatedly by the Commission on Classification and Terminology of the International League Against Epilepsy (1981). This classification, based mainly on the clinical form of the seizure and its electroencephalographic (EEG) features, has been adopted worldwide and is generally referred to as the International Classification[12]. A simpler classification system, another ILAE classification that was developed in 1993 for conducting epidemiological survey on epilepsy. This was named the Epidemiological Classification (EC) and was proposed for only research purposes to overcome technical problems in field studies. [1]

An acute symptomatic seizure is defined as a clinical seizure occurring at the time of a systemic insult or in close temporal association with a documented brain insult. Suggestions are made to define acute symptomatic seizures as those events occurring within 1 week of stroke,

traumatic brain injury, anoxic encephalopathy, or intracranial surgery; at first identification of subdural hematoma; at the presence of an active central nervous system (CNS) infection; or during an active phase of multiple sclerosis or other autoimmune diseases. In addition, a diagnosis of acute symptomatic seizure should be made in the presence of severe metabolic derangements (documented within 24 h by specific biochemical or hematologic abnormalities), drug or alcohol intoxication and withdrawal, or exposure to well-defined epileptogenic drugs

Acute symptomatic seizures were further grouped into two broad categories: 1) acute symptomatic seizure caused by acute neurological insult; and 2) acute symptomatic seizure caused by acute metabolic disorder.

Some of the most common situations are listed

Head injury

Seizures occurring within 7 days of a traumatic brain injury.

Cerebrovascular accident

Seizures occurring within **7** days of any cerebrovascular accident.

CNS infection

Seizures occurring in the course of active CNS infection.

CNS tumor

Seizures occurring as the presenting symptom of a CNS tumor.

Post intracranial surgery

Seizures occurring in the immediate postoperative period of an intracranial neurosurgical intervention

Toxic

Seizures occurring during the time of exposure to recreational drugs (e.g., cocaine), prescription drugs (e.g., aminophylline, imipramine), drug overdose, environmental exposure (carbon monoxide, lead, camphor, organophosphates), and alcohol (acute alcohol intoxication).

Withdrawal

Seizures occurring in association with elimination of alcohol and drugs (e.g., barbiturates, benzodiazepines) .

Metabolic

Seizures related to systemic disturbances, e.g., electrolyte imbalance, hypoglycemia, uremia, cerebral anoxia, and eclampsia.

Fever

Seizures occurring with fever in the absence of CNS infection in children.

Multiple causes

Seizures occurring with several concomitant conditions.

Undefined

Seizures occurring in the context of any acute not otherwise definable condition

Classification of Generalised and partial seizures [12, 13]

I. Generalized onset

A. Seizures with tonic and/or clonic manifestations

1. Tonic-clonic seizures

2. Clonic seizures

3. Tonic seizures

B. Absences

1.Typical absences

2. Atypical absences

3. Myoclonic absences

C. Myoclonic seizure types

1. Myoclonic seizures

2. Myoclonic astatic seizures

3. Eyelid myoclonia

D. Epileptic spasms

E. Atonic seizures

II. Focal onset (partial)

A. **Local**

1. **Neocortical**

a. Without local spread

i Focal clonic seizures

ii Focal myoclonic seizures

iii Inhibitory motor seizures

iv Focal sensory seizures with elementary symptoms

v Aphasic seizures

b. With local spread

i Jacksonian march seizures

ii Focal (asymmetrical) tonic seizures

iii Focal sensory seizures with experiential symptoms

2. Hippocampal and parahippocampal

B. With ipsilateral propagation to:

1. Neocortical areas (includes hemiclonic seizures)

2. Limbic areas (includes gelastic seizures)

C. With contralateral spread to:

1. Neocortical areas (hyperkinetic seizures)

2. Limbic areas (dyscognitive seizures without automatisms [psychomotor])

D. Secondarily generalized

1. Tonic-clonic seizures

2. Absence seizures

3. Epileptic spasms (unverified)

III. Neonatal seizures

Status epilepticus

I. Epilepsia partialis continua (EPC)

A. As occurs with Rasmussen syndrome

B. As occurs with focal lesions

C. As a component of inborn errors of metabolism

II. Supplementary motor area (SMA) status epilepticus

III. Aura continua

IV. Dyscognitive focal (psychomotor, complex partial) status epilepticus

A. Mesial temporal

B. Neocortical

V. Tonic-clonic status epilepticus

VI. Absence status epilepticus

A. Typical and atypical absence status epilepticus

B. Myoclonic absence status epilepticus

VII. Myoclonic status epilepticus

VIII. Tonic status epilepticus

IX. Subtle status epilepticus

Localization-related epilepsies and syndromes

1.1 Idiopathic

Benign childhood epilepsy with centrotemporal spikes

Childhood epilepsy with occipital paroxysms

Primary reading epilepsy

1.2 Symptomatic

Chronic progressive epilepsia partialis continua of childhood (Kojewnikow syndrome)

Syndromes characterized by seizures with specific modes of precipitation

Temporal lobe epilepsies

Frontal lobe epilepsies

Parietal lobe epilepsies

Occipital lobe epilepsies

1.3 Cryptogenic

2. Generalized epilepsies and syndromes

2.1 Idiopathic

Benign neonatal familial convulsions

Benign neonatal convulsions

Benign myoclonic epilepsy in infancy

Childhood absence epilepsy

Juvenile absence epilepsy

Juvenile myoclonic epilepsy

Epilepsy with GTCS on awakening

Other generalized idiopathic epilepsies not defined above

Epilepsies with seizures precipitated by specific modes of activation

2.2 Cryptogenic or symptomatic

West syndrome

Lennox-Gastaut syndrome

Epilepsy with myoclonic-astatic seizures

Epilepsy with myoclonic absences

2.3 Symptomatic

2.3.1 Nonspecific etiology

Early myoclonic encephalopathy

Early infantile epileptic encephalopathy with suppression burst

Other symptomatic generalized epilepsies not defined above

2.3.2 Specific syndromes

Epileptic seizures may complicate many disease states. Under this heading are included diseases in which seizures are a presenting or

predominant feature

3. Epilepsies and syndromes undetermined whether focal or generalized

3. I With both generalized and focal seizures

Neonatal seizures

Severe myoclonic epilepsy in infancy

Epilepsy with continuous spike-waves

Acquired epileptic aphasia (Landau-Kleffner syndrome)

Other undetermined epilepsies not determined above

3.2 Without unequivocal generalized or focal features.

All cases with generalized tonic clonic seizures in which clinical and EEG findings do not permit classification as clearly generalized or localization related, such as in many cases of sleep grand mal (GTCS) are considered not to have unequivocal generalized or focal features.

4. Special syndromes

4.1 Situation-related seizures

Febrile convulsions

Isolated seizures or isolated status epilepticus

seizures occurring only when there is an acute metabolic or toxic event due

to factors such as alcohol, drugs, eclampsia, nonketotic hyperglycemia

Classification of seizures according to risk factors

Epileptic seizures and the epilepsies may be a manifestation of many cerebral or systemic diseases. The first step in categorization of seizures should be based on the presence or absence of a presumed acute precipitating insult, which will permit distinction into provoked and unprovoked seizures. Provoked seizures are therefore equivalent to acute symptomatic or situation-related seizures. Single or recurrent unprovoked seizures may belong to two possible categories: symptomatic seizures or epilepsies (of presumed remote cause) and seizures or epilepsy of unknown causes. Identification of the cause may depend on the degree of investigation, which also depends on availability of ancillary tests.

3.1 Symptomatic seizures or epilepsies – consequence of a known cerebral dysfunction

3.1.1 Acute symptomatic – seizures are in close temporal association (within 7 days) with an acute systemic, metabolic or toxic insult and with acute CNS insult (infection, stroke, cranial trauma, intracerebral hemorrhage, acute alcohol intoxication or withdrawal).

3.1.2. Unprovoked seizures

Seizures may occur in relation to a well demonstrated antecedent condition, substantially increasing the risk for epileptic seizures. Two

major subgroups may be categorized: Remote symptomatic unprovoked seizures owing to conditions resulting in a static encephalopathy. Such cases are individuals with epilepsy subsequent to an insult to the CNS, such as infection, cerebral trauma, or cerebrovascular disease, which are generally presumed to result in a static lesion. Second subgroup is due to symptomatic unprovoked seizures owing to progressive CNS disorders

Evaluation of a first seizure [8,14,15]

History of the event

A description of the circumstances surrounding a paroxysmal event can provide important diagnostic clues. A witnessed, 90-second episode that involved loss of consciousness, stiffening, and jerking of the extremities followed by muscle soreness, headache, and the need to sleep for several hours afterwards strongly suggests a tonic-clonic seizure

KEY ELEMENTS IN HISTORY

Before the event

Unusual stress (eg, severe emotional trauma)

Sleep deprivation, Recent illness

Unusual stimuli (eg, flickering lights)

Use of medications and drugs

Activity immediately before event (e.g., change in posture, exercise)

During the event

Symptoms at onset (e.g., aura)

Temporal mode of onset: gradual versus sudden

Duration: brief (ictal phase <5 min) versus prolonged

Stereotypy: duration and features of episodes nearly identical versus changing

Time of day: related to sleep or occurring on awakening

Ability to talk and respond appropriately

Ability to comprehend

Ability to recall events during the seizure

Abnormal movements of the eyes, mouth, face, head, arms, and legs

Bowel or bladder incontinence

Bodily injury

After the event

Confusion

Lethargy

Abnormal speech

Focal weakness or sensory loss (i.e., Todd's paralysis)

Headache, muscle soreness, or physical injury

Past medical history

A review of the events leading up to the seizure may reveal factors that suggest it was provoked. Causes of provoked seizures include alcohol withdrawal, substance abuse, hypoxia, fever, electrolyte imbalance, hypoglycemia, and sleep deprivation.

Drug history

Theophylline, meperidine hydrochloride , isoniazid , antipsychotic drugs (especially clozapine and phenothiazines), radiocontrast dyes, alkylating agents, and ß-lactam antibiotics, quinolones are among the most commonly implicated medications in seizure.

Physical examination

A thorough physical examination can help uncover possible causes of a seizure. Findings may include evidence of trauma, infection, malignancy, congenital anomalies, and prior neurologic events (e.g., focal weakness, spasticity suggesting previous stroke).

During an emergency department evaluation of a patient immediately after a seizure, vital signs should be measured and a general medical examination performed. Guidelines for physical examination are as follows:

- Examine the patient for injuries from the seizure or fall.
- Check oxygen saturation and auscultate the chest for possible aspiration.
- Measure heart rhythm and rate, blood pressure, and orthostatic changes for assessment of syncope.
- Auscultate for carotid murmurs or carotid bruits and sources of embolic stroke.
- Check for rapid pulses, which are often present after seizure and may help in evaluation of psychogenic seizures.

An electrocardiogram should be obtained to identify cardiac rhythm, detect possible ischemia, and measure the QT interval. Prolonged QT syndrome often presents with simple or convulsive syncope. Electrocardiography and 24-hour ambulatory continuous electrocardiographic (Holter) monitoring can help identify cardiac arrhythmias. The possibility of a recent myocardial infarction should be considered, particularly in elderly patients, in whom myocardial infarction may occur from the stress of a seizure.

Neurologic examination

The purpose of the neurologic examination is to identify focal or diffuse cerebral dysfunction. This information is particularly helpful in

localization-related epilepsy. The presence of various features offers clues to the focus of a seizure. For example, aphasia suggests a left frontal, temporal, or parietal onset. Right or left hemiparesis suggests foci from the contralateral motor cortex.

In initial evaluation of a seizure, patients should be observed for fluency of language, facial asymmetry, gaze preferences, and pupillary asymmetry. The last presents in patients who have herniation from brain swelling caused by parenchymal or epidural bleeding and in those who have a rapidly growing brain tumor. The presence of pronator drift may indicate subtle weakness not detected by strength testing. Sensory deficits suggest parietal lobe dysfunction. An extensor plantar response may be noted for some time after a seizure and is not necessarily a pathologic finding.

Diagnostic testing

Laboratory workup is an essential part of evaluation of seizure. Measurement of glucose, calcium, magnesium, thyroid hormone, and liver enzyme levels, as well as toxicology screening (including blood alcohol levels), may reveal common medical causes of seizures. A complete blood cell count may suggest infection, anemia, or sickle cell disease.

In patients suspected to have had an infection or a fever or to have exhibited abnormal behavior just before the event, lumbar puncture should be performed after assessment of the possible risks of the procedure (e.g., coagulopathy, mass lesion). Patients who are immune compromised because of corticosteroid use, recent transplantation, or HIV infection should undergo cerebrospinal fluid evaluation to detect possible fungal, bacterial, or viral infection. In patients with a systemic malignant condition, cytologic evaluation of cerebrospinal fluid can identify meningeal carcinoma.

Electroencephalogram:

- EEG should be performed within 24 hours of the seizure because it is significantly more sensitive when obtained during that period [16]. If the routine EEG findings are normal, a sleep-deprived EEG should be performed.

- Standard EEG detects epileptiform discharges in 29% of patients. Standard EEG combined with sleep-deprived EEG shows epileptiform discharges in 48% of patients [17].

- Schreiner and Pohlmann[18] studied the value of an EEG taken within 48 hours of the first seizure in an adult. They found that 38.0% of patients without seizure recurrence had normal EEGs, while only

10.2% of patients with seizure recurrence had normal EEGs. Focal epileptiform activities were found significantly more frequently (26.5% vs. 13.0%) in patients with seizure recurrence than in patients without seizure recurrence.

Limitation of EEG:

An estimated 0.4% of adults and 2.8% of children who have never had a seizure may have interictal epileptiform discharges. Furthermore, a normal EEG does not refute the diagnosis of epilepsy. The initial EEG reveals epileptiform activity in only 40% of the patients with probable epilepsy.

Imaging studies

The role of imaging studies depends on the stage of evaluation. Immediately after a seizure, computed tomography can detect the presence of bleeding or gross structural lesions. However, magnetic resonance imaging is the study of choice because it is more sensitive and specific for evaluating structural lesions and brain parenchyma. Particular attention should be directed to the hippocampus for evaluation of lesions (e.g., mesial temporal sclerosis) and to the cortical architecture for detection of abnormalities (e.g., dysplasia).

Incidence of Acute Symptomatic Seizures

Overall Incidence

Only two studies provide detailed information regarding the incidence of acute symptomatic seizures [19,20,21] The incidence of seizures occurring at the time of systemic metabolic insults or temporally associated with an insult to the central nervous system (CNS) was determined for the residents of Rochester, Minnesota. The age-adjusted incidence rates for 1955 to 1984, the period of most complete case ascertainment, was 39 per 100,000 person-years (U.S. 1970 population). This rate is somewhat higher than that reported in Gironde, Bordeaux, France [21] which was 29 per 100,000 person-years.

Gender

From the conditions associated with acute symptomatic seizures, men seem to be at higher risk than women. In both the French study[21] and the U.S. study[19], the age-adjusted incidence in men was considerably higher than that in women.

Age

The age-specific incidence of acute asymptomatic seizures in the U.S. study[19] was by far the highest during the first year of life . This is attributable to the high incidence of acute symptomatic seizures associated

with metabolic, infectious, and encephalopathic causes during the neonatal period. Incidence declined in childhood and the early adult years and reached a nadir of 15 per 100,000 person-years among those 25 to 34 years of age. After 35 years of age, the incidence increased progressively, and reached 123 per 100,000 among those older than 75 years of age. Cerebrovascular disease accounted for about half of all acute symptomatic seizures in persons older than 65 years of age. As mentioned previously, age-adjusted incidence is considerably higher in men than women. The sex difference was greatest at the extremes of age. Between the ages of 15 and 44, there were few differences among all causes of acute symptomatic seizures. Seizures associated with eclampsia in women of childbearing age offset a lower incidence of seizures among women associated with other causes. The age-specific incidence rates of acute symptomatic seizures in the French[21] and U.S.[19] studies were similar in middle-aged men, but the U.S. rates were higher in children younger than 15 years of age and in the elderly.

Causes of Acute Symptomatic Seizures

The major causes of acute symptomatic seizures in both the French and U.S. studies were traumatic brain injury, cerebrovascular disease, drug withdrawal, and CNS infection. The underlying causes of the seizures are reflected in patterns of age, sex, and time period. Although the overall

proportional distribution by cause was similar in the United States and France, no age or etiology specific data are available for the latter study.

Acute Symptomatic Seizures Associated with Primary Brain Insults

Central Nervous System Infection

Acute symptomatic seizures in infections of the central nervous system are those occurring during the acute phase of infection. About 5% of people with CNS infection can be expected to experience an acute symptomatic seizure.[22] Infections of the CNS accounted for about 15% of all acute symptomatic seizures in both Bordeaux, France,[21] and Rochester, Minnesota.[19] In the latter study, the age-specific trends of acute symptomatic seizures associated with CNS infection followed the pattern of incidence of CNS infection.[23] The highest incidence was during the first year of life and in children younger than 15 years of age; the incidence was lower in adults. Overall, the age-adjusted incidence in men is twice that in women. Seizures are second only to headache as a presenting symptom of neurocysticercosis. It is assumed that most of these seizures are acute symptomatic and are associated with the response to the acute inflammatory response associated with transitional cyst degeneration.[24] Unlike most time delimited insults associated with acute symptomatic seizures, the inflammatory response may last from weeks to months, thus

requiring a modification of some of the usual concepts of acute symptomatic seizures.[25]

Brain Trauma

Seizures occurring within the first week of a traumatic brain injury are generally assumed to be acute symptomatic. Seizures occurring after that time are generally considered late or unprovoked, although it would certainly be more appropriate to include the concept of stabilization in such definitions. In civilian studies of traumatic brain injuries, about 6% of all cases are associated with acute symptomatic seizures.[26,27] The frequency of early seizures increases with severity of injury and probably represents a surrogate for severity. Acute symptomatic seizures associated with head trauma accounted for about 15% of all acute symptomatic seizures occurring in Rochester[19] and only about 5% of all cases in Bordeaux[21]. Traumatic acute symptomatic seizures in Rochester were more common in men (age-adjusted rate, 8.6) than in women (age-adjusted rate, 4.8) at all ages. The age specific incidence of head trauma is trimodal.[28,29] Given similar levels of severity of injury, children are at higher risk for acute symptomatic seizures than adults.[26]

Cerebrovascular Disease

Following the example of brain injury, acute symptomatic seizures associated with cerebrovascular disease are generally limited to seizures occurring within 1 week of the acute ictus. Between 5% and 10% of individuals with a cerebrovascular insult experience a seizure at the time of stroke.[30,31,32] The frequency varies with the nature of the insult and is probably highest in those with intracerebral haemorrhage. In Bordeaux, cerebrovascular disease accounted for about one third of all cases of acute symptomatic seizures, compared with only 15% of all cases in Rochester. Paralleling the incidence of cerebrovascular disease,[33] acute symptomatic seizures associated with stroke are rare in persons younger than 55 years of age; the incidence rises rapidly with increasing age, reaching 54.6 per 100,000 among persons older than 75 years of age. The age-adjusted incidence of acute symptomatic seizures is higher in men than in women (9.4 vs. 4.7 per 100,000 cases).[33] The sex-specific difference is particularly dramatic in those 65 to 74 years of age: 55.1 per 100,000 in men versus 15.5 per 100,000 in women. In Rochester, the age-specific incidence of acute symptomatic seizures associated with cerebrovascular disease was stable from 1945 through 1974 but fell in the final decade along with the incidence of stroke.[33] The age-adjusted incidence fell through the years

from 8.6 in the decade 1955 to 1964 to 6.5 in the decade 1964 to 1974 to 5.1 in the decade 1975 to 1984.

Brain Tumor

There is debate about how to classify seizures associated with brain tumors. A high proportion of individuals with brain tumors may experience acute symptomatic seizures.[34] In Rochester, acute symptomatic seizures associated with primary or secondary brain tumors occurred at all ages but were rare in persons younger than 45 years of age. The age-specific incidence rates in persons older than 45 years of age were constant at 6 to 8 per 100,000 person-years. Unlike most other acute symptomatic seizures, seizures associated with neoplasm were equally common in men and women.

Acute Symptomatic Seizures Associated with Systemic Disturbances

Seizures occurring in association with systemic insults are easy to identify, although strict operational definitions are elusive. The severity of a systemic insult or the timing of the seizures from the start of the insult is seldom specified.[35] Nonetheless, this category accounts for a substantial proportion of all acute symptomatic seizures.

Eclampsia

In the Rochester studies[36], 15 women had seizures attributed to eclampsia during a 50-year period; this accounted for about 3% of acute symptomatic seizures in women, an incidence of 2.8 per 100,000 women ages 15 to 44 years.. In Houston, Texas, the incidence of eclampsia in two inner-city hospitals from 1982 to 1992 was 0.7 per 1,000 deliveries, a frequency similar to that in Rochester, Minnesota, in the 1940s.

Toxic Insults

Seizures may occur in association with a variety of toxic insults (e.g., carbon monoxide poisoning or acetylsalicylic acid overdose). Toxic insults accounted for about 5% of all acute symptomatic seizures in Rochester, but only a small proportion of cases in Bordeaux. This may be a consequence of differences between these two studies in inclusion criteria for this category. In Rochester, the overall incidence of acute symptomatic seizures associated with toxic insults was 2.2 per 100,000 cases, with the highest incidence in the elderly (older than 75 years of age).

Drug Withdrawal

Most drug withdrawal seizures are associated with abuse of ethanol and, less frequently, barbiturates or other substances. This category of acute symptomatic seizures accounted for about 15% of cases in

Rochester[19] and about one third of cases in Bordeaux.[21] Incidence for this group of acute symptomatic seizures peaked in the 35- to 54-year-old age group. Withdrawal seizures were the primary cause of acute symptomatic seizures for those 25 to 55 years of age. The incidence of seizures attributed to this cause progressively increased during the study period.

Metabolic Insults

Systemic metabolic illness accounted for about 10% of all acute symptomatic seizures in Rochester[19] and about 15% of cases in Bordeaux.[21] In Rochester, the incidence of acute symptomatic seizures attributed to metabolic insults was highest during the first year of life. This was largely caused by hypocalcemia or hypoglycemia in newborns. These cases were identified primarily before 1960; they are virtually nonexistent at the present time in this community, although it is conceivable that they continue to account for a substantial proportion of newborn cases in less medically sophisticated areas. Seizures associated with metabolic disturbances were rare between the second week of life and 55 years of age. There was a slight rise in incidence after this age.

Prognosis of Acute Symptomatic Seizures

In general, acute symptomatic seizures are a reflection of disease severity and, as such, are associated with a relatively high mortality

rate.[37,38,39] However, prognosis obviously varies with the underlying condition. No studies address the influence of acute symptomatic seizures on risk for mortality within given conditions, so it is impossible to assess the contribution of seizures per se on mortality. In survivors of neurologic insults, those with acute symptomatic seizures seem to be consistently at increased risk for subsequent epilepsy compared with those without acute symptomatic seizures.[40] This is probably not related to a kindling phenomenon but rather to the increased severity of initial insult in those with acute symptomatic seizures. Acute symptomatic seizures associated with metabolic insults are not associated with a similar increase in risk for subsequent epilepsy. None of the cases of eclampsia in the Rochester cohort had subsequent seizures. There has been no systemic study of the risk for epilepsy in people with other metabolic conditions

TREATMENT

The treatment [41]of seizures of all types can be divided into the use of antiepileptic drugs, the surgical excision of epileptic foci and other surgical measures, the removal of causative and precipitating factors, and the regulation of physical and mental activity .Choices of antiepileptic drugs depends on type of seizure and patient characteristics .Preferably start a conventional or first line AED like phenytoin , phenobarbitone, carbamazepine, oxcarbamazepine ,or valproate .Newer AED's are

lamotrigine ,leviteracetam, topiramate, zonisamide, gabapentin, tiagabine and felbamate.

Treatment of the underlying condition along with AED is very important in patients with acute symptomatic seizures .Seizures due to metabolic and withdrawal states, treatment with anticonvulsants is usually not necessary as long as the underlying disturbance is rectified. Antiepileptic drug therapy should be started in any patient with recurrent seizures of unknown etiology or a known cause that cannot be reversed. Initiating therapy in a patient with a single seizure is controversial . Patients with structural brain lesions like tuberculoma, NCC or single enhancing lesion, brain tumor, vascular malformation, or brain abscess need AED until resolution of lesion and maintain on an antiepileptic medication for 6 to 12 months[42], and an attempt is made to withdraw medications only if the patient has been completely seizure-free[(1)]. If seizures are refractory to medication, the patient may benefit from surgical removal of the epileptic brain region. Single seizure after stroke may not be treated and if there is high risk for recurrence AED can be given[43,44,45] . Level A evidence is present only for lamotrigine and gabapentin as first-line antiepileptic drugs for treatment of partial-onset seizure in the elderly [46.] Slow release formulations of carbamazepine (400 mg) have also been found to be equally effective.[47] For patients with CVT use of AED is

recommended for one year.[42]Patients with brain tumors AED should be given before and after surgery . Patients with uremic seizures safer AED‘s are phenobarbitone, lamotrigine,valproate and phenytoin .[42]

Patients with tuberculoma or TB meningitis should be treated with ATT. 6month course is acceptable but should be treated 9-12 months in patients who have an inadequate resolution or positive culture during treatment . Steroids should be given to prevent complications . For brain abscess high dose parenteral antibiotics and surgical drainage are advised . Neurocysticercosis is treated with albendazole 15mg/kg /d in 2 doses for 8 days or praziquantel 50mg/kg/d for 15 days [48]. For patients with CVT anticoagulants initially intravenous followed by oral and antibiotics if it is septic thrombophlebitis are employed . Patients with tumor have to be treated surgically followed by radiotherapy or chemotherapy .

Patients with status epilepticus [49]should be treated promptly as the condition is associated with high mortality. Quick assessment of cardiorespiratory function and airway and insert large-bore intravenous line and draw blood for glucose, blood urea nitrogen, electrolytes, and a metabolic and drug screen. A normal saline infusion is begun and a bolus of glucose is given (with thiamine if malnutrition and alcoholism are factors). Diazepam is given intravenously at a rate of about 2 mg/min until the seizures stop or a total of 20 mg has been given or

lorazepam, 0.1 mg/kg given by intravenous push at a rate not to exceed 2 mg/min . A loading dose 20 mg/kg of phenytoin is administered by iv at a rate of less than 50 mg/min or fosphenytoin at 150 mg/min. If the seizure is not controlled repeat phenytoin 7-10mg/kg.Consider sodium valproate 25mg/kg or phenobarbitone 20mg/kg infusion if the seizure still continues. Admit in ICU and give iv anaesthesia with midazolam or propofol as next step .Once the seizure is controlled continue maintenance AED.

AIM AND OBJECTIVES

To analyse the demography, etiological profile, the pattern of seizures, the clinical findings, usefulness of various investigations, and the incidence of potentially curable causes for the patients presenting with acute symptomatic seizures in the age group of 60 years and above.

MATERIALS AND METHODS

THE STUDY GROUP

The study was conducted on inpatients and out patients of Institute of Neurology, Department of Internal Medicine and Intensive medical care unit of Rajiv Gandhi Govt General Hospital Chennai. Approval from the hospital ethical committee was obtained. The study was observational, cross sectional study in nature designed to analyse the patients in age group of 60 years and above , who presented with acute symptomatic seizures. The sample size was 100 and the study period was from January 2009 to March 2011.

INCLUSION CRITERIA:

1. Patients admitted with first episode of acute symptomatic seizures .
2. Age 60 years and above.
3. Patients admitted for other medical conditions, who developed acute symptomatic seizures during the hospital stay.

EXCLUSION CRITERIA:

1. Patients with previous history of seizures.
2. Idiopathic seizures.
3. History of head injury

Clinical data was collected from patients and witnesses in a systematic manner and added to a database, which included a checklist of seizure antecedents and the symptoms associated with seizure. The first task was to ascertain if at all, the presenting complaint is a seizure. In a few instances, even when the presenting history was ambiguous seizure recurrences were witnessed for confirmation. The clinical diagnosis on the seizure type, whether partial or generalized was made.

In depth probes in the history for provocation factors and features suggesting organicity were attempted. Significant past medical history if any were noted. A thorough clinical examination was performed at the time of admission and relevant findings recorded. A routine metabolic screening, which included blood sugar, urea, serum creatinine, electrolytes and liver function tests (if indicated), were done at the time of admission.

Lumbar puncture and CSF analysis was done if infective etiologies were suspected. Earliest possible EEG was attempted and was performed using 32 channel digital EEG recorder.

CT brain plain in all patients and contrast studies when necessary were done in all patients in the study group. MRI brain was done when indicated. Limitations were encountered in affordability of patients for MRI scanning. Early EEG (within 24hours of onset of seizures) could not be performed due to delay in referral of the patients to this institution and because of the time taken for stabilizing patients.

RESULTS AND ANALYSIS

Acute symptomatic seizures in the age group of 60 years and above were studied. The result of the study was shown below.

EPIDEMIOLOGY

Number of cases of Acute Symptomatic seizures studied in 60 years and above was 100.

Table 1- Age Distribution

	Cases	
	No	**Percentage**
60-70	52	52%
71-80	32	32%
>80	16	16%

In the present study, patients age ranges from 60-87 years. Most of the patients were in the age group of 60-70 years, which was about 52% of the total number of cases.

Table 2-Sex Distribution

Sex	Cases	
	No	**Percentage**
Male	60	60%
Female	40	40%

Out of 100 cases in this study, 60% were male and 40% were females.

Table 3- Type of Seizures

Type of Seizures	Cases	
	No	**Percentage**
Partial	61	61
Generalized	39	39

Out of 100 cases in this study 61% had partial seizures, 39% had generalized seizures. Simple partial seizures were seen in 41% of cases, simple partial seizures with secondary generalization, were seen in 20% of cases. Status epilepticus was seen in 27% of cases.

Table 4- Associated Non Convulsive Symptoms

Symptoms	No
Limb weakness	62
Cranial nerve deficit	30
Speech disturbance	14
Fever	15
Headache	15
Vomiting	14

62% of cases with acute symptomatic seizures with cerebrovascular disease presented with limb weakness, cranial nerve deficit were seen in 30% of cases, speech disturbances were seen in 14% of cases, patients with central nervous system infections and cerebral venous thrombosis had headache and vomiting. 15% of patients had fever at the onset of illness. Headache was present in 15 patients of which 7 of the patient had meningitis.

Table-5 Profile of Significant Past Medical Illness

Past medical illness	No
Diabetes Mellitus	27
Systemic Hypertension	22
Chronic Kidney Disease	3
Pulmonary Tuberculosis	5
Coronary Artery Disease	7
Neoplasm	4

Out of 100 patients, 27 patient had diabetes mellitus, 22 patient had systemic hypertension, 6 patients had both diabetes mellitus and systemic hypertension, 5 patients had past history of pulmonary tuberculosis, 7 patients had coronary artery disease, 3 patients had chronic kidney disease, 4 patients had history of carcinoma, 2 of them had bronchogenic carcinoma, 1 had renal carcinoma and 1 had bladder carcinoma.

Table-6- Profile of Neurological Signs at Admission

Neurological signs	No
Altered sensorium	21
Cranial nerve deficit	16
Motor system deficit	62
Sensory system deficit	6
Signs of meningeal irritation	8

Altered sensorium level was seen in 21 patients in our study which ranges from coma to drowsiness, cranial nerve deficit were seen in 16 patients. Most of the patients had facial nerve involvement, sensory system deficit were seen in 6 patients, signs of meningeal irritation were been in 8 patients.

Table-7- Etiology Profile

Causes	No	Percentage
Cerebrovascular disease	62	62
Central Nervous system infections	15	15
Metabolic causes	10	10
Alcohol withdrawal seizures	5	5
Toxins	4	4
Neoplasm	4	4

Out of 100 patients with acute symptomatic seizures in the study group, cerebrovascular disease were seen in 62 patients which is the most common cause. Central nervous system infections contributed to 15% of patients, metabolic causes were seen in 10 patients, alcohol withdrawal seizures were seen in 5 patients. Toxins and neoplasm induced seizures were seen in 4 patients each.

Table 8- Etiology and Age Distribution

Causes	Age Group		
	60-70	**71-80**	**>80**
Cerebrovascular disease	29	20	13
Central Nervous system infections	7	6	2
Metabolic	6	3	1
Alcohol withdrawal seizures	3	1	1
Toxins	2	1	1
Neoplasm	4	-	-

Out of 100 patients in this study group, 51patients fall in the age group between 60-70 years. 31 patients fall in the age group between 71-80 years and 18 patients in the age group 80 and above. Most of the causes which contributes to seizures in this study occurred in the age group between 60 -70 years of age.

Table-9 -Etiology and Sex Distribution

Causes	Sex	
	Male	**Female**
Cerebrovascular disease	32	30
Central Nervous system infections	9	6
Metabolic	6	4
Alcohol withdrawal seizures	5	-
Toxins	3	1
Neoplasm	4	-

In this study, most of the patients were male, 59 of patients were seen in male and 41 patients were seen in female sex group and all causes of seizures were commonly observed in male gender.

Table10 -Etiology Profile of Cerebrovascular Disease

Type	Number
Ischemic Cerebrovascular disease	39
Intracerebral Haemorrhage	19
Cerebral Venous thrombosis	4

Cerebrovascular disease was the most common cause of acute symptomatic seizures in this study, out of 62% cases with cerebrovascular disease, ischemic cerebrovascular disease, were seen in 39% of patients, Intra cerebral haemorrhage were seen in 19% of patients and cerebral venous thrombosis were seen in 4% of patients.

Table 11 -Etiology Profile of Central Nervous System Infection

Type	Number
Meningitis	8
Encephalitis	4
Tuberculoma	2
Brain Abscess	1

Central nervous system infections was the second most common cause of seizures in this study, which is about 15%, meningitis were seen in 8% of patients and encephalitis were seen in 4% of patients.

Metabolic causes were observed in 10% of cases. Hypoglycemia was the commonest metabolic cause which contributes to 5% of total cases. Other causes being uremia in 3% and hyperglycemia in 2% of total cases. Brain secondaries were seen in 4% of cases . Bronchogenic carcinoma were seen in 2 cases as primary, renal and Bladder carcinoma was seen in 1 each.

Table 12-Etiology Profile of Metabolic Causes

Causes	Number	Percentage
Hypoglycemia	5	5
Hyperglycemia	2	2
Uremia	3	3

Toxins induced causes was seen in 4 patients.organophosphorous poisoning was seen in 3 patients and permethrin poisoning was observed in 1 patients.

Table 13- Etiology Profile of Patient with Status Epilepticus

Causes	Number
Cerebrovascular Disease	15
CNS Infection	6
Alcohol withdrawal	1
Metabolic	3
Toxins	2

Status epilepticus was seen in 27 patients in the study group. Cerebrovascular diseases was the most common cause for status epilepticus in this study. Status epilepticus occurred in 15 patients with cerebrovascular disease,central nervous infection was the next most common cause for status epileptiucs in 6 patients.

Table-14- Profile of EEG in the Study Group

EEG Findings	Number	Percentage
Normal	73	73
Focal Slowing	14	14
Diffuse Slowing	9	9
Epileptiform discharge	4	6

EEG was normal in 73 patients. Out of 23 patients with abnormal EEG, most common abnormality was focal slowing in 14 patients. Diffuse slowing was observed in 9 patients, only 4 patients showed epileptiform discharges.

Table – 15: Profile of EEG in cerebrovascular Disease

EEG Findings	Number
Normal	45
Focal slowing	14
Epileptiform discharge	3

Out of 62 patients with cerebrovacscular disease. EEG was normal in 45 patients. Abnormal EEG was seen in 17 patients.

Table 16-CT brain abnormality in the study group

CT Brain finding	Number
Ischemic Infarction	35
Parenchymal haemorrhage	19
Cortical atrophy	20
Haemorrhagic infarction	2
Tuberculoma	1
Meningeal enhancement	3
Neoplasm	4

CT scan brain was abnormal in 61 patients, Ischemic infarction was seen in 35 patients which was the most common abnormality. Parenchymal haemorrhage was seen in 19 patients. Cortical atrophy was seen in 20 patients, which was found in combination with other abnormal CT findings. Haemorrhagic infarction was seen only in 2 patients. Ring enhancing tuberculoma lesion was seen in only 1 patient.

Table17-New Lesions uncovered in MRI brain

MRI Brain finding	Number
Ischemic Infraction	4
Encephalitis	4
Cerebral venous thrombosis	2
Tuberculoma	2
Cerebral abscess	1

MRI brain was done in 75 cases. MRI was useful in uncovering, lesions which were missed in CT brain in 13 patients. 4 patients with ischemic infarction who had normal CT brain findings, showed abnormality in MRI brain. Similarly 4 patients with encephalitis showed abnormality in MRI brain.

DISCUSSION

Rajiv Gandhi Government General Hospital, Chennai is the tertiary referral hospital and is the premier institute in the state of Tamil Nadu. Various cases have been referred from Government sector hospitals like primary health centres, Government General Hospitals, District head quarters hospitals and many private hospitals, not only from nearby Chennai but also from nearby districts in Tamil Nadu. Study on acute symptomatic seizures in the elderly patients was conducted from the period of January 2009 to March 2011, which included 100 patients.

Epidemiology

Analysing the age group in this study, the maximum incidence of acute symptomatic seizures occurred in the age groups of 60-70 years. This was about 52% in our study. In the study conducted by Ruggers et al[50] on retrospective study of seizures in the elderly maximum incidence was in the age group of 60-70 years, which was about 44%. In another study conducted by Annergers et al[19] on incidence of acute symptomatic seizures in the Rochester, the maximum incidence of 45% was in the age group of 60-70 years.

The study group comprised of 60% males and 40% females. Most authors report small to moderate preponderance of men in their studies of

acute symptomatic seizures in the elderly individuals. In the study conducted by Hauser et al[19], the incidence of acute symptomatic seizures was 53% in males and 47% females.

Table18-Comparison of sex distribution with other study

Sex	Our study %	Hauser et al %
Male	60%	53%
Female	40%	47%

Etiology

Most common cause of seizures in our study was cerebrovascular disease, followed by central nervous system infections and metabolic causes. Cerebrovascular disease contributed to 62% in our study. Central nervous system infection contributed to 15% and metabolic causes contributed to 10%. Alcohol withdrawal seizures, toxins and neoplasm contributed to remaining 13% of cases.

In the study conducted by Hauser et al[19]on acute symptomatic seizures, cerebrovascular disease contributed to 57% , metabolic causes contributed to 11%, central nervous system infection in 3%, toxin induced causes in 9%, Alcohol withdrawal seizures in 8% and neoplasm in 12% of cases.

In another study conducted by Alana Holt-Seitz et al[51] on seizures in the elderly, etiology and prognosis, cerebrovascular disease contributed to 46% of cases, metabolic causes contributed to 20%,central nervous system infections in 12% of cases, neoplasm in 17% of cases and alcohol withdrawl seizures in 5% of cases.

Table19-Comparison of etiology profile with other studies

	Hauser et al%	**Alana Holt etal %**	**Our study%**
Cerebrovascular disease	57	46	62
Central nervous system infections	3	12	15
Metabolic causes	11	20	10
Alcohol withdrawl seizures	8	5	5
Toxins	9	–	4
Neoplasm	12	17	4

Cerebrovascular disease was the most common cause for seizures in our study, which was seen in 62% of cases, when compared with various other studies, cerebrovascular disease was the most common cause for acute symptomatic seizures in the elderly, similar to our study. It was 57%

in the study conducted by Hauser et al[19], 46% in the study conducted by Alana Holt[19], 52% in the study conducted by Ang Rt et al[52], 44% in the study conducted by Wood Cock[53], 73% in the Fine et al [54] and 57% in the study conducted by Gupta et al[55].

Table 20-Comparison of cerebrovascular disease as etiology with other studies

Study	Percentage
Hauser et al	57
Alana Holt et al	46
Ang RT et al	41
Wood cock et al	44
Fine et al	73
Gupta et al	40
Our Study	62

As observed in various other studies, cerebrovascular disease was the most common cause for seizures. Of the 62 cases of cerebrovascular disease, 63% of cases were due to Ischemic cerebrovascular disease, 31% of cases were due to intracerebral haemorrhage and 6% of cases were due to cerebral venous thrombosis.

In the study conducted by Daniel et al[56] on prevalance and predictors of the early seizures and status epilepticus after first stroke, Ischemic infarction contributed to 60% of cases, intracerebral haemorrhage to 30% of cases and subarachnoid haemorrhage in 10% of cases. Thus ischemic infarction was the most common etiology, similar to our study.

Table 21-Comparison of cerebrovascular disease profile with other studies

Cerebrovascular Disease	Daniel et al	Our study
Ischemic infarction	60	63
Intracerebral haemorrhage	30	31
Subarachnoid haemorrhage	10	–
Cerebral venous thrombosis	–	6

Central nervous system infections, contributed to 15% of etiology of seizures in our study, meningitis is the most common cause among infections, which contributed to 8% of total 15% of cases. In the study conducted by Alana Holt etal[51]on seizures in elderly, incidence of central nervous system infection was 15% and in other study conducted by Hauser et al[19], the incidence was 3% of total cases.

Table 22-Comparison of central nervous system infection as Etiology with other studies

Study	Percentage
Alana Holt et al	15
Hauser et al	2
Our study	15

Metabolic causes contributed to 10% of cases as etiology in our study. Among the 10% of cases, hypoglycemia was the common cause, which was seen in 5% of cases, uremia in 3% of cases and hyperglycemia in 2% of cases. They were the most readily treatable cause for acute symptomatic seizures, especially those patients detected to have hypoglycemic seizures. Hence a review at the metabolic parameters at admission was mandatory and when detected was most rewarding for the treating physician.

Alcohol withdrawl seizures contributed to above 5% of cases in our study. History was the most important thing in patients diagnosed to have alcohol withdrawl seizures. In the study conducted by Franson KL et al[57], drug induced seizure in the elderly, alcohol withdrawl seizures was the major cause for seizures.

Neoplasm contributed to 4% of cases in our study. All the 4 cases were due to secondary metastasis, of the 4 cases, 2 cases had bronchogenic carcinoma, 1 patient each had bladder and renal carcinoma. So the patients with known primary carcinoma presenting with seizures, the first thing which should be ruled out as the cause for seizures is intracranial metastasis.

As already discussed, most of the cases occurred in the age group between 60-70 years, which was 52% of total cases. Among 52 % of cases in 60-70 years age group, cerebrovascular disease contributed to 30%, central nervous system infection in 7%, metabolic causes in 6%, Alcohol with drawl in 3% ,toxins in 2% and neoplasm in 4% of cases.

Seizure type

The seizure type in our study, classified as per international league against epilepsy, revised classification of epileptic seizured, revealed partial seizure in 61% and generalized seaizures in 31% of cases.

In the study conducted by Chung Yung et al[58], on epileptic seizures in the elderly people, etiology and seizure type, partial seizures was observed in 80% and generalized tonic clonic seizures in 20% .Similarly in the study conducted by Eugene Ramsay et al[3], showed partial seizures in 73% and generalized tonic clonic seizures in 27% and in the study

conducted by Hauser et al, showed partial seizures in 52% and generalized tonic clonic seizures in 48%. The observation of seizure type in the above mentioned studies showed preponderance of partial seizures, which was similar to our study.

Table23-Comparison of seizure type with other studies

Seizure type	Our Study	Hauser et al	Chung Yung et al	Ramsay et al
Partial	61	80	52	73
Generalized	39	20	48	27

Among the seizure type observed in cerebrovascular disease patients, partial seizures was observed in 82% of patients and generalized tonic clonic convulsions in 18%. In the study conducted by Daniel et[56] al on prevalence and predictors of early seizures and status epilepticus after first stroke, partial seizures was seen in 59% of cases and generalized tonic clonic convulsions in 41% of cases.

Table 24-Comparison of seizures in cerebrovascular disease with other study

Seizure type	Daniel et al	Our Study
Partial	59	82
Generalized	41	18

Incidence of status epilepticus was seen in 27% of the patients in our study. In the study conducted by Hauser et al[59] on status epilepticus and associated morbidity, status epilepticus was observed in 30% of cases in acute symptomatic seizures in the elderly group.

In the study conducted by Sung et al[60] on status epilepticus in the elderly the incidence of status epilepticus was 30% and similarly in the study on incidence of status epilepticus in Rochester minnestoa by Hesdorffer et al[61] the incidence of status epilepticus was 35% in acute onset symptomatic seizures in the elderly group. Among the 27% of status epilepticus in our study cerebrovascular disease, contributed to 15% of cases, central nervous system infections in 6% cases ,metabolic causes in 3% of cases, toxin induced causes in 2% and alcohol withdrawl seizures in 1% of cases.

Table 25-Comparison of status epilepticus incidence with other studies

Status epilepticus	**Percentage**
Hauser et al	30
Sung et al	30
Hesdorffer et al	35
Our study	27

Among the cerebrovascular disease who presented with seizures status epileptcius was seen in 24% of patients.Similarly in the study conducted by Daniel et al[56] on status epilepticus in stroke, the incidence of status epilepticus was 27% of total cases.

Table 26-Comparsion of status epilepticus in cerebrovascular disease with other study.

Status epilepticus	**Percentage**
Daniel et al	27
Our study	24

Among the non convulsive symptoms, limb weakness was the most common symptom reported by the attenders during admission. It is

obvious as the most common cause of seizures in our study group was stroke which was 62%. Fever was present in 15 cases, most of the patients had central nervous system infections. It is important here to emphasize that fever is one of the provocative factor for seizures. Headache was present in 15 patients most of the patient presented with headache had meningitis.

Previous history of diabetes mellitus was present in 27 patients, out of them 2 patients presented with hypoglycemia induced seizures and 2 patients with hyperglycemia induced seizures. Seizures due to uremia occurs in patient with previous history of chronic kidney disease in 3 patients. Systemic hypertension and diabetes mellitus occurred in 20 patients, which is significant risk factor for cerebrovascular disease. Among the 4 patient with seizures due to neoplasm, 2 patients had presented with history of bronchogenic carcinoma and 1 patient each of bladder and renal cell carcinoma ,it is well known fact that the bronchogenic, bladder and renal carcinoma has prediction for haematogenous spread into the brain.

EEG was done in 100 patients in our study group. Abnormal EEG was found in 27 patients. The average period from the onset of seizures to the recording of EEG was six days. The late referral was due to the time taken for the patient stabilization before shifting the patient to EEG room.

The yield would have been better if we done earlier.The most common abnormality in EEG was focal slowing in 14 patient, diffuse slowing in 9% cases. Epileptiform discharges was seen in only 4 patients.

CT scan brain was done is all patients in the study group, in which ischemic infarction was seen in 35 cases, intracerebral haemorrhage in 19 cases,tuberculoma in 1 case,haemorrhagic infarction in 2 cases, and hydrocephalus in 1 case. Cortical atrophy was seen in 15 patients which was found in combination with other abnormality. In our study ischemic infarction was the most common abnormal findings in CT brain. In the study conducted by Alan Holt et al[51] on seizures in elderly in etiology and prognosis the most common CT brain finding was ischemic infarction which was observed in 46% of patients.

MRI brain was done in 75 cases. MRI was useful to identify lesions which were missed in CT brain in 13 cases. The most common abnormality which was detected in MRI, which were missed in CT brain were 4 cases each of ischemic infarction and encephalitis, 2 cases of tuberculoma and cerebral venous thrombosis were also detected in MRI brain which was missed in CT brain. MRI was also very useful in improving the details of CT findings.

SUMMARY

A study on acute symptomatic seizures in the elderly was a cross sectional study of 100 patients done in the Rajiv Gandhi Government General Hospital, Chennai. The maximum incidence of seizures occurred in the age group of 60-70 years which was about 52%. There was preponderance of occurrence of seizures in males, which was about 60% of total cases. The commonest etiology in the study was cerebrovascular disease, which contributed to 62% in our study. The second most common cause was central nervous system infections which contributed to 15%. Among the cerebrovascular disease, ischemic infarction was the most commonest cause, which was observed in 63% of total cerebrovascular disease patients. Partial seizures was the most common seizure type encountered in our study which was seen in 61%. Status epilepticus was seen in 27% of the patients in our study. Limb weakness was the most common non convulsive symptom reported. Motor system abnormality was the most common total neurological deficit, which was observed in 62 patients. EEG was abnormal in 27 patients. Ischemic infarction was the most common CT Brain abnormality, detected in our study, which was seen in 35 patients. MRI Brain was useful in detecting new lesions in 13 patients which were missed initially in CT Brain.

CONCLUSION

- The maximum incidence of seizures occurred in the age group of 60 to 70 years in the study.

- Cerebrovascular disease was the most common etiology observed in our study. Among cerebrovascular disease, ischemic cerebral infarction was the most common cause. Central nervous system infections, was the second most common cause followed by metabolic causes in our study. So all efforts should be taken to evaluate the patients who presented with seizures in the elderly patients to identify the etiology, for accurate management.

- Partial seizures was the most common seizure type encountered in the study.

- Immediate non contrast CT brain was useful, for emergency patients, presenting with seizures to guide appropriate acute management. MRI Brain was useful in detecting lesions, which were missed in CT Brain. Both CT Brain and MRI Brain were indispensable in patients with acute symptomatic seizures in the elderly.

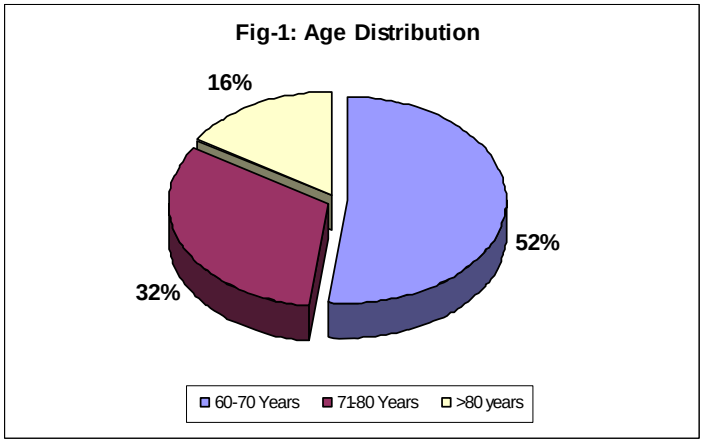


Fig-1: Age Distribution
16%
52%
32%
60-70 Years
71-80 Years
>80 years

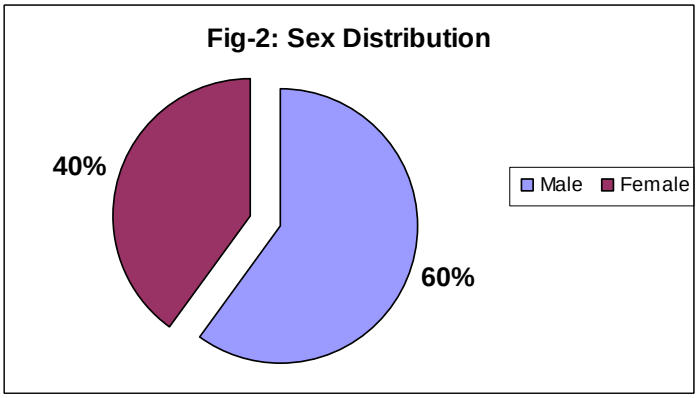


Fig-2: Sex Distribution
40%
60%
Male
Female

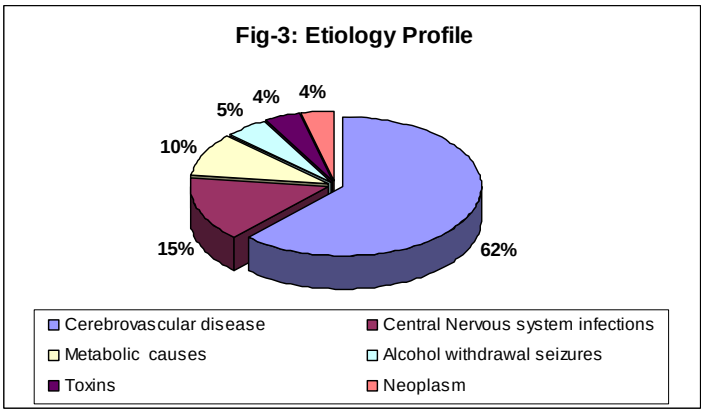


Fig-3: Etiology Profile
4%
4%
5%
10%
15%
62%
Cerebrovascular disease
Central Nervous system infections
Metabolic causes
Alcohol withdrawal seizures
Toxins
Neoplasm

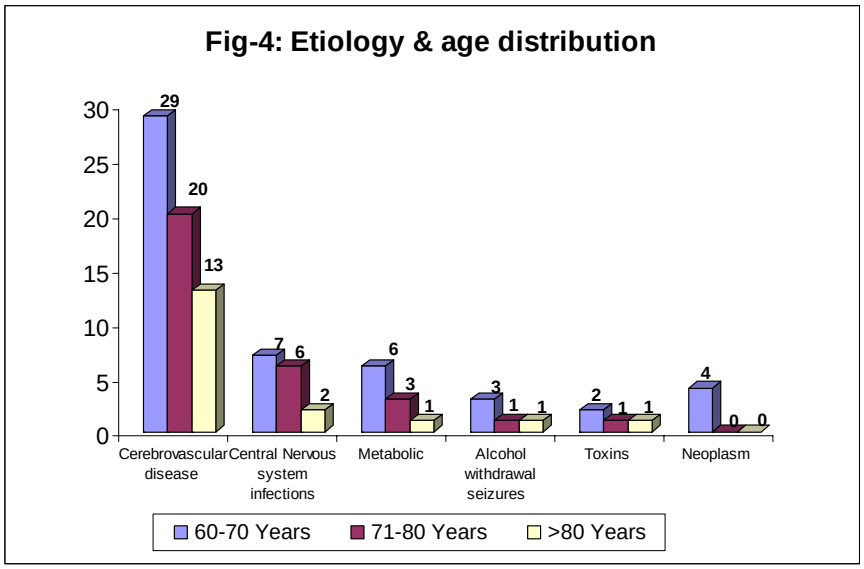


Fig-4: Etiology & age distribution
30
25
20
15
10
5
0
29
20
13
7
6
2
6
3
1
3
1
1
2
1
1
4
0
0
Cerebrovascular disease
Central Nervous system infections
Metabolic
Alcohol withdrawal seizures
Toxins
Neoplasm
60-70 Years
71-80 Years
>80 Years

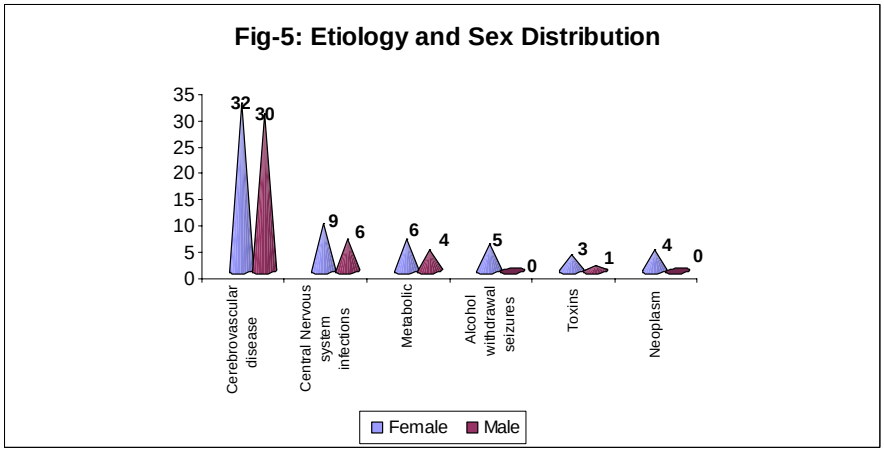


Fig-5: Etiology and Sex Distribution
35
30
25
20
15
10
5
0
32
30
9
6
6
4
5
0
3
1
4
0
Cerebrovascular disease
Central Nervous system infections
Metabolic
Alcohol withdrawal seizures
Toxins
Neoplasm
Female
Male

Fig- 6-Etiology Profile of Cerebrovascular Disease

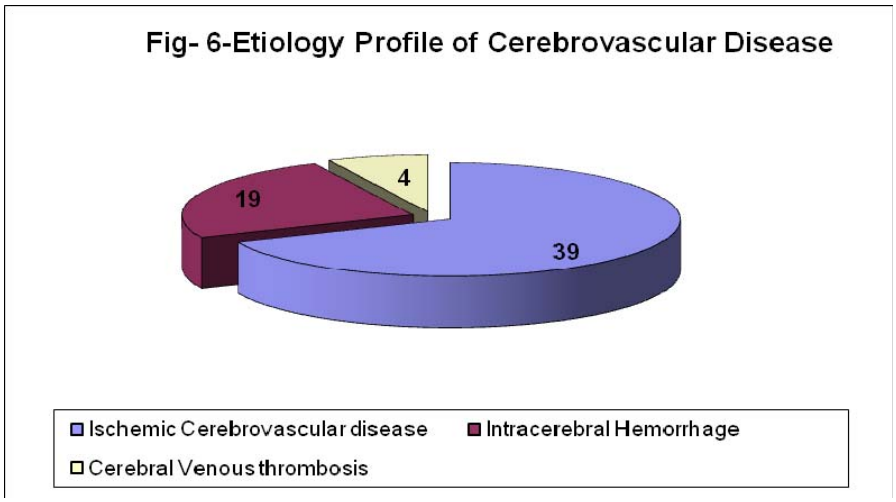


Fig-7: Etiology Profile of Central Nervous System Infection

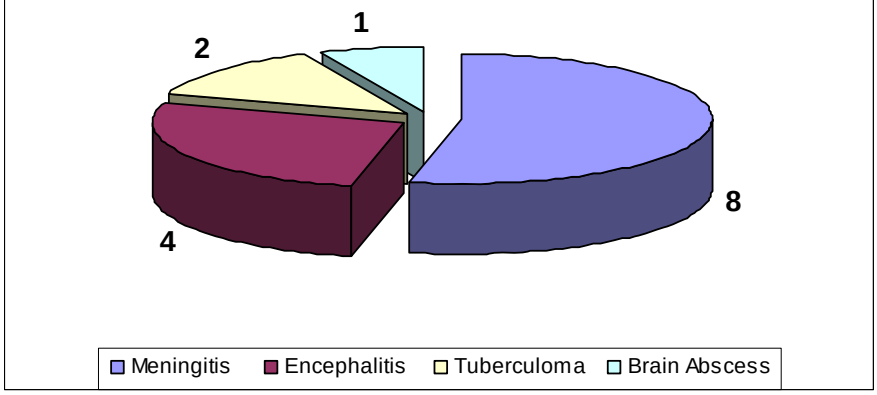


Fig-8: Etiology Profile of Patient with Status Epilepticus

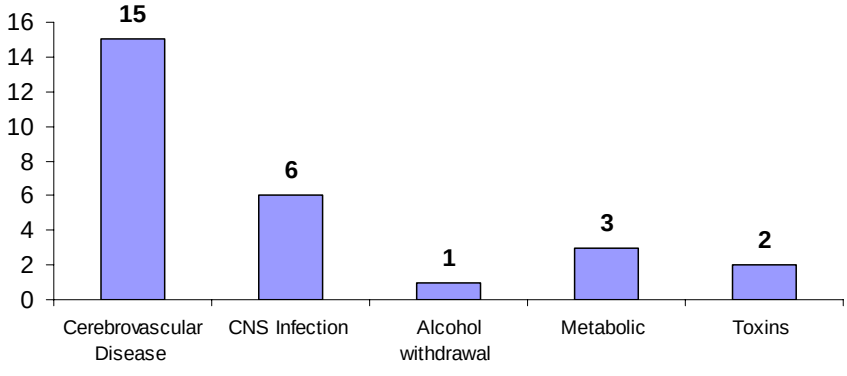


Fig -9 EEG WITH BILATERAL EPILEPTIFORM ACTIVITY

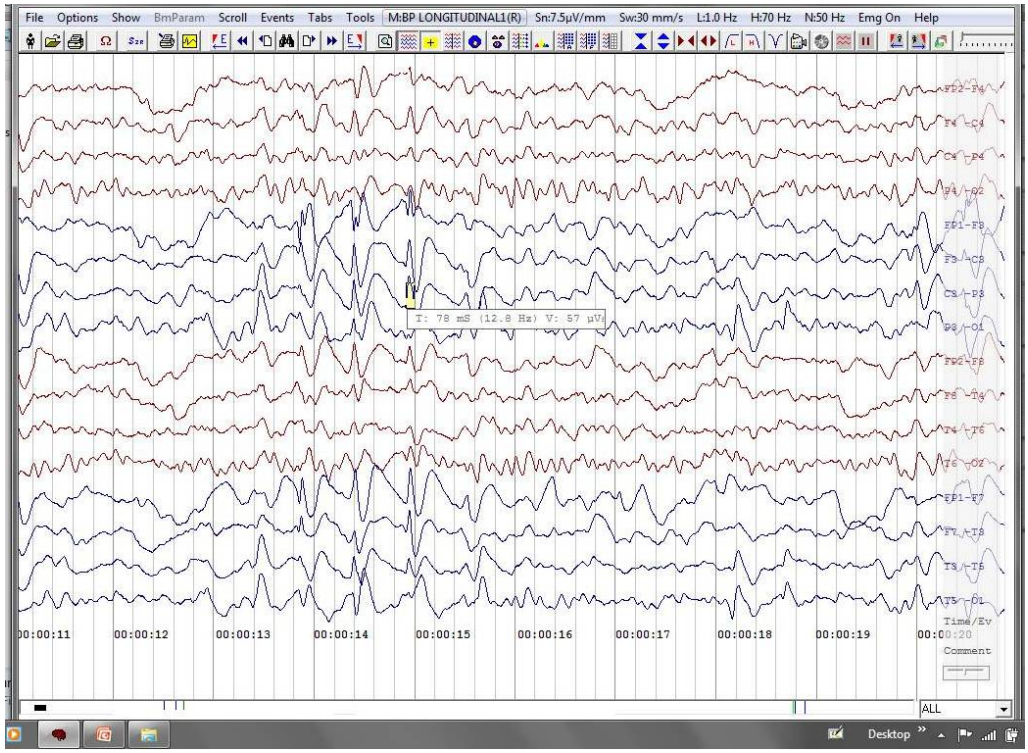


Fig -10 EEG WITH GENERALISED SLOW WAVES

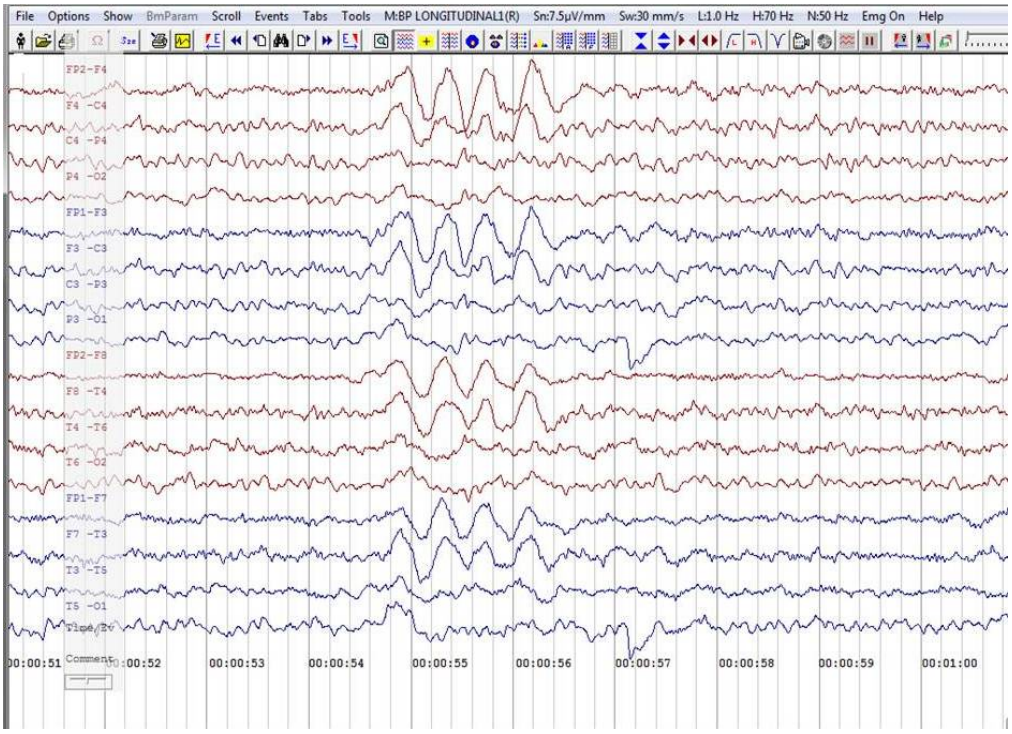


Figure-11: MRI Brain- GRE
Intracerebral Haemmorhage

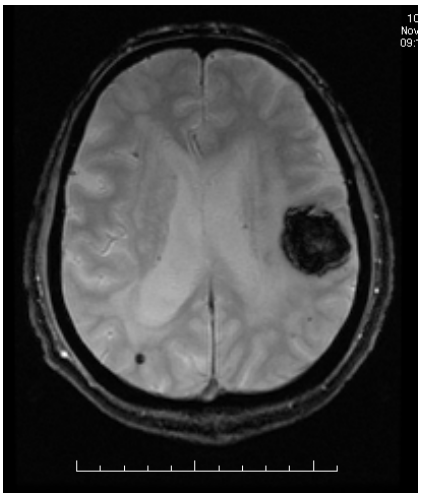


Figure-12 MRI Brain- DWI
Ischemic Infarction

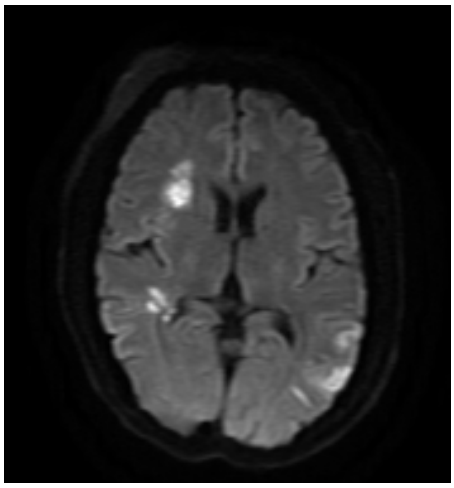


Figure-13: CT Brain
Cortical Vein Thrombosis

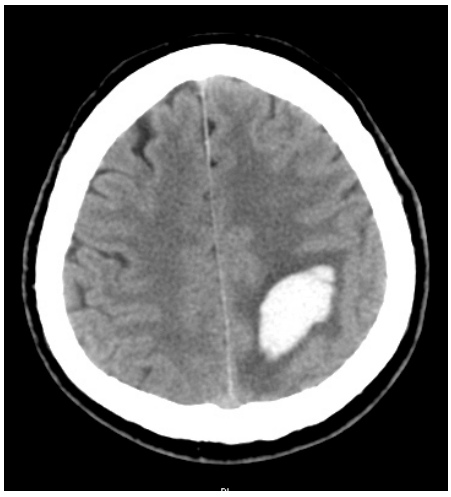


Figure-14:MRI Brain
cerebral metastasis

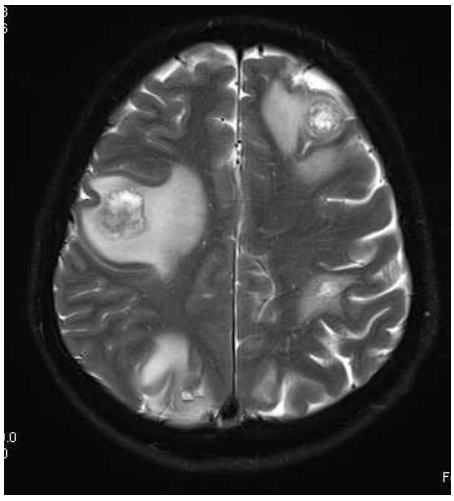


Figure-15: MRI Brain- T1 Contrast Tuberculoma Brain

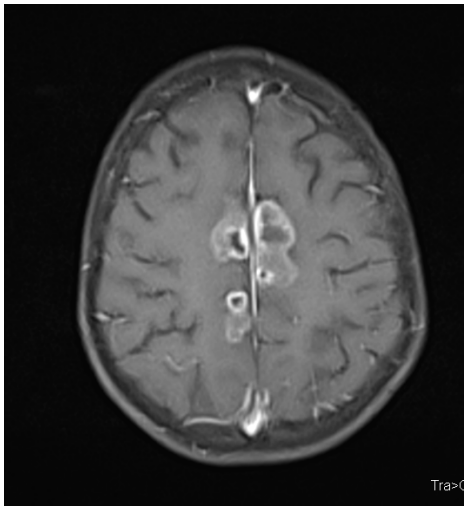
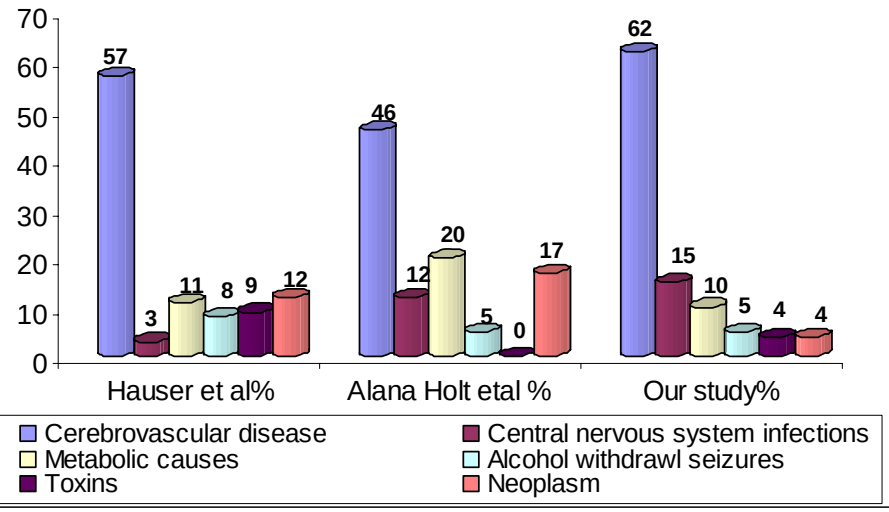


Figure-16: MRI Brain- T2 Flair Herpes Encephalitis

Fig – 17: CT Brain Meningitis

Fig-18: Comparison of etiology profile with other studies



REFERENCES

1. Guidelines for Epidemiologic Studies on Epilepsy, Commission on Epidemiology and Prognosis, International League Against Epilepsy, Epilepsia, 34(4):592-596, 1993

2. Glauser T, Ben-Menachem E, Bourgeois B, Cnaan A, Chadwick D, Guerreiro C, et al. ILAE treatment guidelines: Evidence-based analysis of antiepileptic drug efficacy and effectiveness as initial monotherapy for epileptic seizures and syndromes. Epilepsia 2006;47:1094-120.

3. R. Eugene Ramsay, MD; A. James Rowan, MD; and Flavia M. Pryor, RN, Special considerations in treating the elderly patient with epilepsy Neurology 2004;62(Suppl 2):S24–S29

4. Hauser W.A.,Annegers J. F., Kurland L. T. Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota : 1935-1984. Epilepsia, 1993, 34 : 453-468.

5. Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised classification of epilepsies and epileptic syndromes.Commission on Classification and Terminology of the International League Against Epilepsy. Epilepsia ,1989,30(4):389–399

6. Pohlmann-Eden B, Beghi E, Camfield C, Camfield P. The first seizure and its management in adults and children. British Medical Journal. Feb 11 2006;332(7537):339-42.

7. Hesdorffer D, Benn E, Cascino G, Hauser W. Is a first acute symptomatic seizure epilepsy? Mortality and risk for recurrent seizure. Epilepsia. 2009;50(5):1102-1108

8. Hopkins A, Garman A, Clarke C. The first seizure in adult life, Value of clinical features, electroencephalography, and computerised tomographic scanning in prediction of seizure recurrence. Lancet. 1988;1(8588):721-6

9. Musicco M, Beghi E, Bordo B: Randomized clinical trial on the efficacy of antiepileptic drugs in reducing the risk of relapse after a first unprovoked tonic-clonic seizure. First Seizure Trial Group Neurology 1993 Mar; 43: 478-83

10. Fauci,Braunwald,Hauser,Harrison's Principles of Internal medicine,17 th Edition,Volume II : 2502-2503

11. Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised clinical and electroencephalographic classification of epileptic seizures. From the Commission on Classification and Terminology of the

International League Against Epilepsy. Epilepsia 1981;22(4):489–501

12. Engel J Jr; International League Against Epilepsy (ILAE). A proposed diagnostic scheme for people with epileptic seizures and with epilepsy: report of the ILAE Task Force on Classification and Terminology. Epilepsia 2001;42(6):796–803.

13 . Engel J Jr. Report of the ILAE classification core group. Epilepsia 2006;47(9):1558–1568.

14. Moore-Sledge CM. Evaluation and management of first seizures in adults. Journal of American Family Physician-Sep 15 1997;56(4):1113-20.

15. Tardy B, Lafond P, Convers P, et al. Adult first generalized seizure: etiology, biological tests, EEG, CT scan, in an ED. American journal of Emergency Medicine. Jan 1995;13(1):1-5

16. King MA, Newton MR, Jackson GD, et al. Epileptology of the first-seizure presentation:a clinical,electroencephalographic,and magnetic resonance imaging study of 300 consecutive patients. Lancet. Sep 21998;352(9133):1007-11

17. van Donselaar CA, Schimsheimer RJ, Geerts AT, Declerck AC. Value of the electroencephalogram in adult patients with untreated idiopathic first seizures. Arch Neurology. Mar 1992;49(3):231-7.

18. Schreiner A, Pohlmann-Eden B. Value of the early electroencephalogram after a first unprovoked seizure. Clin Electroencephalography. Jul 2003;34(3):140-4

19. Annegers JF, Hauser WA, Lee JR-J, et al. Incidence of acute symptomatic seizures in Rochester, Minnesota, 1935-1984. Epilepsia. 1995;36:327-333.

20. Hauser WA, Annegers JF, Kurland LT. Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota: 1935-1984. Epilepsia. 1993;34:453-468.

21. Loiseau J, Loiseau P, Guyot M, et al. Survey of seizure disorders in the French Southwest. I. Incidence of epileptic syndromes. Epilepsia. 1990;31:391-396

22. Annegers JF, Hauser WA, Beghi E, et al. The risk of unprovoked seizures after encephalitis and meningitis. Neurology. 1988;38:1407-1410.

23. Nicolosi A, Hauser WA, Beghi E, et al. Epidemiology of central nervous system infections in Olmsted County, Minnesota, 1950-1981. J Chronic Disorder. 1986;154:399-402

24. Carpio A, Escobar A, Hauser WA. Cysticercosis and epilepsy: a critical review. Epilepsia. 1998;39:1025-1040

25. Carpio A, Hauser WA. Prognosis for seizure recurrence in patients with newly diagnosed neurocysticercosis. Neurology. 2002;59(11):1730-1734

26. Annegers JF, Grabow JD, Groover RV, et al. Seizures after head trauma: a population study. Neurology. 1980;30:683-689.

27. Salazar AM, Jabbari B, Vance SC, et al. Epilepsy after penetrating head injury. I. Clinical correlates: a report of the Vietnam Head Injury Study. Neurology. 1985;35:1406-1414

28. Annegers JF, Grabow JD, Kurland LT, et al. The incidence, causes, and secular trends of head trauma in Olmsted County, Minnesota, 1935-1974. Neurology. 1980;30:912-919.

29. Cooper KD, Tabaddor K, Hauser WA, et al. The epidemiology of head injury in the Bronx. Neuroepidemiology. 1983;2:70-81.

30. Labovitz DL, Hauser WA, Sacco RL. Prevalence and predictors of early seizure and status epilepticus after first stroke. Neurology. 2001;57:200-206.

31. Olsen TS, Hogenhaven H, Thage O. Epilepsy after stroke. Neurology. 1987;37:1209-1211.

32. Viitanen M, Ericksson S, Asplund K. Risk of recurrent stroke, myocardial infarction and epilepsy during long-term follow-up after stroke. European Neurology. 1988;28:227-231

33. Broderick JP, Phillips SJ, Whisnant JP, et al. incidence rates of stroke in the eighties: the end of the decline in stroke? Stroke. 1989;20:577-582.

34. Franceschetti S, Battagha G, Lodrini, et al. Relationship between tumors and epilepsy. In: Brogie G, ed. The Rational Basis of the Surgical Treatment of Epilepsies. London: John Libbey; 1988

35. Decaux G, Soupart A. Treatment of symptomatic hyponatremia. American Journal of Medical Sciences. 2003;326:25-30

36. Hauser WA, Annegers JF, Kurland LT. Prevalence of epilepsy in Rochester, Minnesota: 1940-1980. Epilepsia. 1991;32:429-445

37. Hesdorffer DC, D'Amelio M. Mortality in the first 30 days following incident acute symptomatic seizures. Epilepsia. 2005;46(Suppl 11):43-45.

38. Lhatoo SD, Johnson AL, Goodridge DM, et al. Mortality in epilepsy in the first 11 to 14 years after diagnosis: multivariate analysis of a long-term, prospective, population-based cohort. Ann Neurology. 2001;49(3):336-344.

39. Loiseau P, Loiseau J, Picot MC. One-year mortality in Bordeaux cohort: the value of syndrome classification. Epilepsia. 2005;46(Suppl 11):11-14.

40. Hesdorffer DC.Logroscino G, Cascino G, et al. Risk of unprovoked seizure after acute symptomatic seizure: effect of status epilepticus. Ann Neurology. 1998;44(6):908-912.

41. Fauci,Braunwald,Hauser,Harrison's Principles of Internal medicine,17 th Edition,Volume II : 2507-2511

42. Usha Kant Misra, Jayantee Kalita Management of provoked seizures Annals of Indian Academy of Neurology 2011;14:2-8

43. Marson A, Jacoby A, Johnson A, Kim L, Gamble C, Chadwick D;et al. Immediate versus deferred antiepileptic drug treatment for early

epilepsy and single seizures: A randomised controlled trial. Lancet 2005;365:2007-13.

44. Chandra B. First seizure in adults: To treat or not to treat, Clinical Neurology Neurosurgery, 1992;94:S61-3.

45. Ryvlin P. When to start antiepileptic drug treatment: Seize twice might not harm. Current Opinion Neurology 2006;19:154-6

46. Glauser T, Ben-Menachem E, Bourgeois B, Cnaan A, Chadwick D, Guerreiro C, et al. ILAE treatment guidelines: Evidence-based analysis of antiepileptic drug efficacy and effectiveness as initial monotherapy for epileptic seizures and syndromes. Epilepsia 2006;47:1094-120

47. Saetre E, Perucca E, Isojärvi J, Gjerstad L. An multicenter doubleblind double-dummy randomised trial comparing lamotrigine andslow-release carbamazepine for treating newly diagnosed epilepsyin the elderly. Proceedings of the 7th European Congress of Epileptology; 2006. p. 3.

48. Del Brutto OH, Roos KL, Coffey CS, Garcia SH. Meta analysis: Cysticidal drugs for neurocysticercosis: Albendazole and praziquantel. Ann Internal Medicine 2006;145:43-51

49. Shorvon S, Baulac M, Cross H, et al. The drug treatment of status epilepticus in Europe: consensus document from a workshop at the first London Colloquium on Status Epilepticus. Epilepsia 2008;49(7):1277–1285.

50. Kevin H. Ruggles, Sharon M. Haessly, and Richard L. Berg et al Prospective Study of Seizures in the Elderly in the Marshfield Epidemiologic Study Area Epilepsia, 42(12):1594–1599, 2001

51. Alana Holt-Seitz, Elaine C. Wirrell and Mecheri B. Sundaram Seizures in the Elderly: Etiology and Prognosis Canadian Journal of Neurological Sciences. 1999; 26: 110-114

52. Ang RTetal, Utterback RA. Seizures with onset after forty years of age; role of cerebrovascular disease. South Med J 196639: 1404-8.

53. Woodcock S etal, Cosgrove JBR. Epilepsy after age 50. Neurology 1964; 14:34-40.

54 . Fine W etal . Epileptic syndromes in the elderly. Gerontology Clinics l966;8: 121-33

55. Gupta K etal , Epilepsy in the elderly. How far to investigate. European Journal of Clinical Practise 1983;37:259-62

56. Daniel L. Labovitz, MD, MS; W. Allen Hauser, MD; and Ralph L. Sacco, MD, MS Prevalence and predictors of earlyseizure and status epilepticus after first stroke Neurology 2001;57:200–206

57. Franson KL, Hay DP, Neppe V, et al. Drug-induced seizures in the elderly. Causative agents and optimal management. Drugs Aging 1995; 7:38- 48.

58. Chung-yang sung and Nai-shin chu Epileptic Seizures in Elderly People Aetiology and Seizure Type , Oxford Journals of Medicine , Age and Ageing Volume19, Issue1 Pp. 25-30

59. Hauser WA, Cascino GD, Annegers JF, Rocca WA. Incidence of status epilepticus and associated mortality [abstract]. Epilepsia 1994; 35(suppl 8):33

60. Sung CY, Chu NS. Status epilepticus in the elderly: etiology, seizure type and outcome. Acta Neurology Scandanivia. 1989;80:51-56

61. Hesdorffer DC, Logroscino G, Cascino G, et al. Incidence of status epilepticus in Rochester, Minnesota, 1965–84. Neurology. 1998;50:735–741.

PATIENT CONSENT FORM

STUDY TITLE : A STUDY ON ACUTE SYMPTOMATIC SEIZURES IN THE ELDERLY

Study Centre : Madras Institute of Neurology,

Madras Medical College, Chennai – 600 003

Patient's Name :

Patient's Age :

Identification Number : Patient may check (✓) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethics committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and to faithfully co-operate with the study team, and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this Study on acute symptomatic seizures in the elderly ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological and urine examination. ☐

Signature / Thumb Impression _____ Place _____ Date _____

Patient's Name and Address: _____

Signature of the Investigator : _____ Place _____ Date _____

Study Investigator's Name : _____

PROFORMA FOR EVALUATION

Name **Age** **Sex**

Address

Occupation **Contact no**

op/ip no **ward** **DOA** **DOD**

min no **EEG no**

Referred from

ELABORATION OF SEIZURE

Witnesse of seizure **Reliability**

BEFORE THE EPISODE

Recent illness (headache / fever)

Unusual stress

Medications

Last alcohol intake

Last meal

Sleep deprivation

Activity just before seizure

DURING THE EPISODE

Time of day

Aura

Duration

Ability to talk & comprehend

Ability to recall events

Movements of eyes face arms legs

Tongue bite frothing

Bowel / bladder incontinence

Bodily injuries sustained

AFTER EVENT

Confusion duration

Focal neurological deficits

Headache

Any other significant symptoms

SIGNIFICANT PAST HISTORY

Diabetic : yes / no duration & treatment

Hypertension CAD CKD

tuberculosis

any others

alcohol intake y / n duration freq quantity last intake

smoking

family h/o seizures

CLINICAL EXAMINATION

General exam

Neuro cut markers

Vitals : BP Pulse RR Temp

CENTRAL NERVOUS SYSTEM EXAMINATION

at presentation Time after seizure

signs of meningial irritation

higher functions

cranial nerves

motor system

sensory system

cerebellum

CVS : RS: P/A :

COURSE DURING HOSPITAL STAY

INVESTIGATIONS :

Hematology TC : DC: P L E B HB : ESR:

Biochemistry

sugar urea creatinine Na k Ca

CSF analysis

Others :

ECG :

Xray Chest:

ECHO :

CT BRAIN :

MRI BRAIN :

EEG :

Treatment

S.No	Name	Age	Sex	Seizures Type	Status epilepticus	Past History	fever	headache	Altered sensorium	FND	B. sugar	B. urea	S .creatinine	EEG	CT Brain	MRI Brain	Diagnosis
1.	Ravi	67	M	P	2	1	2	2	1	1	A	N	N	A	A	A	ICH
2.	Anandhan	71	M	p	1	0	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
3.	Parthiban	81	M	P	2	2,4	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
4.	Durairaj	63	M	G	1	3	2	2	2	1	A	N	N	A	A	A	Ischemic Infarction
5.	Vellaisamy	76	M	G	2	O	2	2	2	1	N	N	N	N	A	A	ICH
6.	Ananthy	61	FM	G	1	7	2	2	2	1	N	N	N	A	A	A	Ischemic Infarction
7.	Anjali	77	FM	P	2	0	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
8.	Vellaiyan	66	M	P	2	3	2	2	2	1	A	N	N	N	A	A	ICH
9.	Gajendran	67	M	P	2	0	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
10.	Eswari	84	FM	P	2	2,4	2	2	2	1	N	N	N	N	A	A	ICH
11.	Kalaiarasi	69	FM	P	2	7	2	1	2	1	N	N	N	N	A	B	CVT
12.	Jayapal	76	M	G	1	1	2	2	2	1	A	N	N	A	A	A	Ischemic Infarction
13.	Kuppusamy	60	M	P	2	4	2	2	2	1	N	N	N	N	A	A	ICH
14.	Ganesan	72	M	P	2	2	2	2	1	1	N	N	N	A	A	A	Ischemic Infarction
15.	Megalai	64	F	G	2	3	2	2	2	1	A	N	N	N	A	A	Ischemic Infarction
16.	Nalini	79	F	P	2	1	2	1	2	1	A	N	N	A	N	A	CVT
17.	Ismail	67	M	P	1	0	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
18.	Chinnasamy	66	M	P	2	2	2	2	2	1	N	N	N	A	A	A	ICH
19.	Bhuvana	75	F	P	2	1	2	2	2	1	A	N	N	N	A	A	Ischemic Infarction
20.	Malar	63	F	G	1	2	2	2	2	1	N	N	N	A	N	A	Ischemic Infarction

S.No	Name	Age	Sex	Seizures Type	Status epilepticus	Past History	fever	headache	Altered sensorium	FND	B. sugar	B. urea	S .creatinine	EEG	CT Brain	MRI Brain	Diagnosis
21.	Andiappan	83	M	P	2	4	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
22.	Malliga	76	F	P	2	3	2	2	2	1	A	N	N	N	A	A	ICH
23.	Rajendran	64	M	P	2	0	2	2	1	1	N	N	N	N	A	A	Ischemic Infarction
24.	Arasi	82	F	G	1	7	2	1	2	1	N	N	N	A	N	A	CVT
25.	Kannan	65	M	P	2	0	2	2	2	1	N	N	N	N	A	A	ICH
26.	Padmini	84	F	G	2	3	2	2	2	1	A	N	N	N	A	A	Ischemic Infarction
27.	Nazir	69	M	P	2	0	2	2	2	1	N	N	N	N	A	A	ICH
28.	Chellammal	83	M	P	2	0	2	2	2	1	N	N	N	N	A	A	ICH
29.	Chellam	61	F	P	2	1	2	2	2	1	A	N	N	N	A	A	Ischemic Infarction
30.	Muthammal	82	F	P	2	0	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
31.	Kumar	63	M	P	1	1	2	2	2	1	A	N	N	A	A	A	Ischemic Infarction
32.	Dhanapal	62	M	P	2	0	2	2	1	1	N	N	N	N	A	A	Ischemic Infarction
33.	Bharani	79	F	P	2	2	2	2	2	1	N	N	N	N	A	A	ICH
34.	Mani	67	M	P	2	1	2	2	2	1	A	N	N	N	A	A	Ischemic Infarction
35.	Kavitha	77	F	P	1	4	2	2	2	1	N	N	N	A	A	A	Ischemic Infarction
36.	Devanayagi	83	F	G	2	2	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
37.	Ramalingam	65	M	P	2	0	2	2	2	1	N	N	N	N	A	A	ICH
38.	Ravichandran	76	M	P	2	1	2	2	2	1	A	N	N	A	A	A	Ischemic Infarction
39.	Ponnammal	67	F	P	2	0	2	2	2	1	N	N	N	A	A	A	Ischemic Infarction
40.	Rani	74	F	P	2	0	2	2	2	1	N	N	N	N	A	A	ICH

S.No	Name	Age	Sex	Seizures Type	Status epilepticus	Past History	fever	headache	Altered sensorium	FND	B. sugar	B. urea	S .creatinine	EEG	CT Brain	MRI Brain	Diagnosis
41.	Sampantham	68	M	P	1	3	2	2	2	1	A	N	N	A	A	A	Ischemic Infarction
42.	Prabakaran	85	M	P	2	0	2	2	2	1	N	N	N	N	N	A	Ischemic Infarction
43.	Chamundi	86	F	P	2	2	2	2	2	1	N	N	N	N	A	A	ICH
44.	Manoharan	78	M	P	2	0	2	2	2	1	N	N	N	N	A	A	CVT
45.	Palanivel	70	M	P	2	1	2	2	2	1	A	N	N	A	A	A	Ischemic Infarction
46.	Lakshmi	87	F	G	2	4	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
47.	Valli	73	F	P	1	0	2	2	1	1	N	N	N	N	N	A	Ischemic Infarction
48.	Bannariammal	63	F	p	2	2	2	2	2	1	N	N	N	N	A	A	ICH
49.	Chinnammal	86	F	P	2	0	2	2	2	1	N	N	N	N	A	A	ICH
50.	Ranganayaki	72	F	P	1	1	2	2	2	1	A	N	N	N	A	A	Ischemic Infarction
51.	Srinivasan	62	M	P	2	0	2	2	2	1	N	N	N	A	A	A	Ischemic Infarction
52.	Bagiyam	61	F	P	2	2	2	2	2	1	N	N	N	A	A	A	Ischemic Infarction
53.	Thiyagarajan	77	M	P	2	1	2	2	2	1	A	N	N	N	A	A	Ischemic Infarction
54.	Gunamani	76	F	P	1	4	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
55.	Govindhan	63	M	P	2	0	2	2	1	1	N	N	N	A	N	A	Ischemic Infarction
56.	Thulasi	82	F	P	2	1	2	2	2	1	A	N	N	N	A	A	ICH
57.	Shanmugam	76	M	P	1	2	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
58.	Harikrishnan	77	M	P	2	1	2	2	2	1	A	N	N	A	A	A	Ischemic Infarction
59.	Gnanamalar	79	F	P	1	0	2	2	2	1	N	N	N	N	A	A	ICH
60.	Panneselvam	65	M	P	2	1	2	2	2	1	A	N	N	A	A	A	Ischemic Infarction

S.No	Name	Age	Sex	Seizures Type	Status epilepticus	Past History	fever	headache	Altered sensorium	FND	B. sugar	B. urea	S .creatinine	EEG	CT Brain	MRI Brain	Diagnosis
61.	Velammal	66	F	P	2	0	2	2	2	1	N	N	N	N	A	A	Ischemic Infarction
62.	Kandhan	68	M	P	2	0	2	2	2	1	N	N	N	N	A	A	ICH
63.	Chinnappan	62	M	P	2	1,7	1	1	2	2	A	N	N	N	A	A	Tuberculoma
64.	Ayyavu	71	M	G	1	0	1	1	1	2	N	N	N	A	N	A	Encephalitis
65.	Kumaresan	61	M	G	2	7	1	1	1	1	N	N	N	N	A	A	Meningitis
66.	Aruna	74	F	G	1	0	1	2	1	2	N	N	N	A	N	A	Encephalitis
67.	Komathi	73	F	G	2	2	1	1	1	1	N	N	N	N	N	B	Meningitis
68.	Vimala	65	F	P	2	7	1	1	1	2	N	N	N	N	N	A	Tuberculoma
69.	Pavai	82	F	G	1	0	1	1	1	2	N	N	N	A	A	A	Meningitis
70.	Alagiri	66	M	G	2	0	1	1	1	2	N	N	N	N	N	B	Meningitis
71.	Dhanapal	67	M	P	2	1	1	1	1	1	A	N	N	N	N	A	Brain Abscess
72.	Ramasamy	84	M	G	1	0	1	1	1	2	N	N	N	A	A	A	Meningitis
73.	Chitra	74	F	G	2	0	1	1	1	2	N	N	N	A	N	B	Meningitis
74.	Loganathan	67	M	G	1	2	1	2	1	2	N	N	N	N	N	A	Encephalitis
75.	Subramanian	76	M	G	2	0	1	1	1	2	N	N	N	N	N	B	Meningitis
76.	Rangammal	68	F	G	1	0	1	1	1	1	N	N	N	N	N	B	Meningitis
77.	Sampath	73	M	G	2	0	1	2	1	2	N	N	N	A	N	A	Encephalitis
78.	Murugan	63	M	G	1	2,5	2	2	1	2	N	A	A	N	N	B	Uremia
79.	Senthil	67	M	P	2	1	2	2	2	2	A	N	N	A	N	B	Hypoglycemia
80.	Ponni	66	F	G	2	1	2	2	2	2	A	N	N	N	N	B	Hypoglycemia

S.No	Name	Age	Sex	Seizures Type	Status epilepticus	Past History	fever	headache	Altered sensorium	FND	B. sugar	B. urea	S .creatinine	EEG	CT Brain	MRI Brain	Diagnosis
81.	Kodhai	72	F	G	2	1	2	2	2	2	A	N	N	N	N	B	Hyperglycemia
82.	Gandhi	76	F	G	2	2,5	2	2	2	2	N	A	A	N	N	B	Uremia
83.	Kumaravel	82	M	G	1	1	2	2	2	2	A	N	N	N	N	B	Hyperglycemia
84.	Chandrasekar	65	M	P	2	0	2	2	2	2	N	N	N	N	N	B	Hypoglycemia
85.	Vadivu	66	F	G	2	2,5	2	2	2	2	N	A	A	N	N	B	Uremia
86.	Palaniappan	64	M	P	1	1	2	2	2	2	A	N	N	N	N	B	Hypoglycemia
87.	Kalairavan	73	M	G	2	0	2	2	2	2	N	N	N	N	N	B	Hypoglycemia
88.	Sivaraman	63	M	G	1	0	2	2	2	2	N	N	N	N	N	B	Alcohol withdrawl seizures
89.	Thangavel	67	M	G	2	0	2	2	2	2	N	N	N	A	N	B	Alcohol withdrawl seizures
90.	Veerannan	77	M	G	2	0	2	2	2	2	N	N	N	N	N	B	Alcohol withdrawl seizures
91.	Ammasi	82	M	G	2	0	2	2	2	2	N	N	N	N	N	B	Alcohol withdrawl seizures
92.	Babu	65	M	G	2	0	2	2	2	2	N	N	N	N	N	B	Alcohol withdrawl seizures
93.	Papathy	65	F	G	1	0	2	2	2	2	N	N	N	N	N	B	OPC poisoning
94.	Soundarajan	72	M	G	2	0	2	2	2	2	N	N	N	A	N	B	OPC poisoning
95.	Nagendran	68	M	G	2	0	2	2	2	2	N	N	N	N	N	B	OPC poisoning
96.	Gopal	82	M	G	2	0	2	2	2	2	N	N	N	N	N	B	Permethrin poisoning
97.	Vanchilingam	63	M	P	2	0	2	2	2	2	N	N	N	A	A	A	Neoplasm
98.	Mohammed	65	M	P	2	0	2	2	2	2	N	N	N	A	A	A	Neoplasm
99.	Palanisamy	68	M	P	2	0	2	2	2	2	N	N	N	N	A	A	Neoplasm
100.	Rajamani	61	M	P	2	0	2	2	2	2	N	N	N	N	A	A	Neoplasm

KEY TO MASTER CHART

SEX

M-male , F-female

SEIZURE TYPE

P-partial seizure , G-generalized seizures

STATUS EPILEPTICUS

1-present , 2-absent

PAST HISTORY

1-diabetes mellitus, 2-systemic hypertension , 3- diabetes mellitus and systemic hypertension , 4-coranary artery disease , 5-chronic kidney disease 6-nepolasm, 7-pulmonary tuberculosis , 0-absent.

FEVER

1-present, 2-absent

HEAD ACHE

1-present, 2-absent

ALTERED SENSORIUM

1-present, 2-absent

FND(focal neurological deficit)

1-present ,2-absent

BLOOD SUGAR

A-Abnormal, N-normal

BLOODUREA

A-Abnormal, N-normal

BLOOD CREATININE

A-Abnormal, N-normal

EEG

A-Abnormal, N-normal

CT BRAIN

A-Abnormal, N-normal

MRI BRAIN

A-Abnormal, N-normal